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The Prevention of Streptococcal Infection of Wounds*

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IF the present conflict resembles that of 1914-18 in any respect whatever, a high proportion of the wounds inflicted will become infected, and these infections of themselves may cause the death or serious disablement of many thousands. Many wounds sustained in civil life are similarly infected. It is therefore of extreme importance that we should consider whether, in the light of the knowledge we now possess, these infections may be prevented.

In general, four varieties of organism infect wounds. Firstly, there are the organisms which cause tetanus, secondly those which cause gas gangrene, thirdly the staphylococci and fourthly the streptococci. The sources of the first three have been known for some time. Both the tetanus and gas gangrene organisms can exist in the form of spores and these may be found with distressing frequency in soil and dirt. It may therefore be assumed that the presence of this undesirable commodity in wounds is fraught with tremendous possibilities. Nor do we need to look very far for the source of the staphylococci because these organisms are invariably present on the skin. But the source of the streptococci has always been a subject on which we had no exact information, although there have been theories in plenty, mostly wrong, to account for their presence in a wound.

The streptococci which are of importance in wounds are almost all those known as haemolytic streptococci or *Str. pyogenes*. The other varieties of streptococci, the viridans and those without action on blood, are for all practical purposes negligible. Now the importance of the haemolytic streptococci as a cause of wound sepsis is not always realized. I cannot give you figures for their

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incidence in the wounds of civil life but I can for wounds of wartime. In the last war, for instance, it was found that no less than 15 per cent of all wounds examined at a casualty clearing station, that is within an average of 12 hours of the infliction of the wound, were already infected with haemolytic streptococci. In another investigation, this time at a base hospital, 23 per cent were infected and in a third investigation on compound fractures of the femur, more than 90 per cent were found to be infected with these organisms. For this reason, haemolytic streptococcal infection of wounds in war is a very serious problem and I have reason to believe that it may be more important in the wounds of civil life than is usually supposed.

It is obvious that if these infections could be prevented we might reduce very considerably the inevitable toll of war and might also reduce the mortality from injuries in civil life. Up to the present we have been unable even to approach this ideal by reason of a number of fundamental difficulties. The most important of these is that hitherto it has not been possible to distinguish pathogenic from non-pathogenic strains of haemolytic streptococci. Perhaps it may surprise you to hear that there are non-pathogenic strains of these organisms. As a matter of fact there are a great many and haemolytic streptococci are, on the whole, fairly widespread in nature. They occur quite frequently on the skin for instance, and although we had always suspected that many of these strains were unable to cause infections in human beings, we were unable to distinguish them from strains which most certainly can. But thanks to the work of Lancefield, we are now able to separate those strains capable of causing infection in human beings from those which cannot. The former are, in bacteriological language, known as group A haemolytic streptococci while those assigned to other groups labelled B to M are almost if not quite harmless for man. Some of these strains, I may mention in passing, are highly pathogenic for lower animals. This test has given us an extremely powerful weapon because we can now take any strain and assess its ability to cause infection in human beings.

Sources of Infection

Employing these methods, let us consider where the organisms causing infection of wounds can have come from. First, we must ascertain whether the organisms can have been implanted in the wound at the time of infliction. This is the explanation for the presence of these organisms which is put forward almost invariably by surgeons. There is, however, a great deal of evidence that this is not the case. Rifle bullets and shell splinters are probably quite sterile while flying through the air because the heat of the propellant would almost certainly kill any organisms on them. Other objects which may cause wounds such as portions of motor car or machinery are almost certainly not infected with these organisms. They cannot live very long in such inhospitable surroundings and even if purposely placed on them would very soon die. I think therefore that we can, in a general way, exonerate the object which inflicts the wound.

The first part of one's person to encounter the object is usually the clothing

through which it passes on its way to the tissues and pieces of cloth may even find their way into the wound. Recently we have been carrying out a very extensive examination of the clothing of ordinary normal individuals and can say quite definitely that the clothing of most of us is not infected with pathogenic strains of haemolytic streptococci. Haemolytic streptococci may be and frequently are present but they are not members of the pathogenic group A. There is, however, an exception. This is the clothing of persons who are so unfortunate as to carry the pathogenic strains in the throat and in these individuals the upper part of the trunk, the pocket handkerchief and the pocket in which it is kept may be infected. The trousers generally escape. The clothing of some carriers is apparently quite uninfected, and in those in which it is infected the organisms are for the most part confined to the areas accessible to the nasopharyngeal secretion. Thus, except for the clothing of some carriers we can, I think, exonerate the clothing.

Very much the same remarks apply to the skin. That of normal individuals may have haemolytic streptococci on it but usually such strains are not members of group A. The same exceptions occur too, in the form of nasopharyngeal carriers whose skin in certain parts of their anatomy may be heavily and persistently infected. These areas include the hair, the skin of the face and the hands but not that of the trunk and legs which are seldom infected. Thus, in general we can acquit the skin.

Now let us consider the dust and the dirt which may find its way into the wound. Nearly everybody thinks that streptococci are present in these substances. But road dust or dirt, the soils of the countryside or the garden are not infected with haemolytic streptococci. We have examined over 50 specimens and while almost any other microbe may be present, pathogenic strains of haemolytic streptococci are certainly not. Tetanus and gas gangrene are undoubtedly due to contamination of the wound with soil but not the particular organisms with which we are dealing. Here again there are exceptions. This is the dust of hospitals in which infected cases are being nursed. This may be heavily infected with pathogenic strains. But not the dust of the road, factory or battlefield.

The only remaining object which finds its way into a wound with any degree of regularity is the air. Exactly as with dust and dirt, the air of the countryside, of factories and well-run hospitals does not contain these organisms. But again there are exceptions. There is now clear evidence that the air of wards in which infected cases are being nursed, scarlet fever and puerperal fever wards in particular, may be and frequently is heavily infected.

Thus the whole of the available evidence suggests that except in rare instances, haemolytic streptococci or pathogenic strains are not present in wounds, even the dirtiest and most contused of them, *at the time of infliction*. If we admit this we must consider what extraneous sources for the organisms are available and the chances that the organisms will reach open wounds.

A fairly extensive survey has been made of recent years of the distribution of pathogenic strains of haemolytic streptococci in nature. As a result of this we know of only two principal reservoirs of these strains, and both of them are

in human beings. The first is the nasopharynx of individuals who are either quite well or are suffering from some variety of nasopharyngeal infection, and may I remind you that many of these individuals have the organisms on their clothing and on their hands as well as in their throats. The second possible source is the infected material coming from suppurating wounds, cellulitis, impetigo, otitis media and similar purulent exudates in other patients in the vicinity of the freshly infected wound.

Let us take the first of these, the individual who has the organisms in his nasopharynx. He may, of course, be suffering from a definite infection such as scarlet fever or tonsillitis which everybody knows is due to these organisms. But haemolytic streptococci do not always produce these characteristic clinical entities so that quite a number of mild sore throats or what pass for common colds may be either due to these organisms or to synergism between them and a virus. There is, furthermore, evidence that the organisms may persist for months after such an infection with their owner quite unaware of it. And finally there is evidence that we may become carriers in the absence of any form of infection. However acquired, we now know that on an average, about seven per cent of normal persons going about their normal daily work carry pathogenic strains of haemolytic streptococci in their nasopharynx. In the majority, the organisms are present on the tonsils and not in the nose, but in some they are in the nose as well. I am inclined to think that the latter are the more dangerous to other people. In passing, I may mention that another ten per cent of the population have haemolytic streptococci in the nasopharynx which are probably not pathogenic.

It may be legitimately asked how these persons coming into contact with an open wound can cause infection in it. Direct experiment has shown that such a carrier, bending over and talking vertically downwards over a blood agar plate can project the organisms on to the medium on which they appear as colonies when it is incubated. Not every carrier can do it, or always do it. Nevertheless a carrier who, unmasked, bends over an open wound and talks—and how seldom do those who attend to wounds remain silent—is evidently running a very great risk of infecting that wound.

But it is not only the breath of the carrier which may directly transmit infection to an open wound, for I have already mentioned that the skin of the face, the hair, the clothing and the hands, particularly the hands, of such a person may be heavily contaminated with pathogenic haemolytic streptococci. If, therefore, he does not prepare his hands with care, or having prepared them, contaminates them by touching himself, he may very easily infect the wound while dressing it.

Now there is every reason to believe that the nasopharyngeal carrier is one of the principal sources from which haemolytic streptococcal infections of all sorts are derived. He has certainly been shown to play an important part in the spread of nasopharyngeal infections such as scarlet fever and tonsillitis, and to be frequently the source of the organisms in puerperal infections. Thus, of a total of 110 puerperal cases studied in four investigations, the source of the

infection was found in no less than 65 of them, to be the nasopharynx of one or other of the attendants at the time of delivery. I have little doubt that a similar study of wound infections would yield very similar results and for this reason I think we must assume that the nasopharynx of a carrier is one of the most important reservoirs from which the organisms are derived.

Let us consider the other reservoir of infection. This, strange as it may appear, is the hospital or home to which the wounded man is sent; or rather, the infected cases already there. These may be discharging purulent wounds, abcesses, cellulitis, whitlows and so on. There may also be cases of otitis media, of puerperal infection or that of ulcerating cancer, all of which may be discharging haemolytic streptococci. Any or all of these may be present in the ward or a neighbouring ward, and sometimes in close proximity to the wounded man. It may well be asked how in a well run hospital, an infection of this type could cause infection in an open wound in another person. There is unfortunately a great deal of evidence that this is perfectly possible. It has, for instance, been recently shown that puerperal infections and those of wounds and burns may pollute the atmosphere about them so that haemolytic streptococci may be isolated from the air. And this in a new, modern hospital embodying all the latest architectural improvements. There is also experimental evidence. It can, for instance, be shown that so long as the infected pus or serous exudate on the dressings of such a case are wet, no dissemination of the organisms into the surrounding atmosphere is likely. If they become dry and remain perfectly still, there is likewise no atmospheric contamination. But let the dried material be agitated in any way, mere folding of a piece of lint infected with a dried serous exudate is sufficient, and large numbers of organisms are immediately released into the atmosphere. Even in an almost imperceptible breeze these particles can be shown to travel at least three metres. In a gale they probably travel far and wide.

This is a danger of which surgeons and nurses seem to be quite ignorant. They will cheerfully nurse infected cases in the same ward as clean, and the astonishing thing is that they usually get away with it. But they do not invariably do so and particularly in the overcrowding and scurry of wartime. For it was shown in the war of 1914-18 that at the base hospital in Etaples in which were collected all the cases of compound fracture of the femur, more than 90 per cent of the wounds were infected with these organisms.

There is also another danger in hospital or home which is far too seldom recognized. In winter it is quite usual for patients in surgical wards to suffer from nasopharyngeal infections. These may be apparently quite trivial but some of them may be connected with haemolytic streptococci. Their presence in a ward along with clean cases is, however, asking for trouble, for these nasopharyngeal infections may also infect the atmosphere about them and so cause infection of wounds in neighbouring beds.

To recapitulate: The organisms present in wounds sustained by carriers may very well have come either from the individual's own throat, his skin or his clothing. But this is certainly not the source of the organisms in most wounds.

We must therefore recognize that the vast majority of haemolytic streptococcal infection of wounds are not due to the injection of the organisms into the raw area at the time of infliction. Infection comes sometime later and from two main sources: (1) from nasopharyngeal carriers who attend to the wound and (2) from other infections in the neighbourhood.

Control Measures

What then can we do? Let us deal with the nasopharyngeal carrier first. Recognition that he may be the source of the organisms shows that anyone who attends a recently inflicted wound, even he who administers first aid on the battlefield or at the roadside, should remember all the time that he may involuntarily be the cause of infection. Ideally, he should wear a mask but of almost equal value is a very simple measure that no one seems to have thought of before. This is merely that he should refrain from speaking (and of course coughing or sneezing) while near the patient or the wound. We do not expel the organisms while merely breathing quietly, it is while speaking, coughing or sneezing that we do so. But if the attendant has any form of nasopharyngeal infection, no matter how trivial, he should, in my opinion, refrain from contact with open tissue until haemolytic streptococci of group A have been proved bacteriologically to be absent from his throat. If this is impossible, and I am quite aware that it often is, he should at least take special pains to eliminate the chances of transfer in the way I have already described. A second point in this connection is that he should take particular care over the toilet of his hands. More often than not the washing process is carried out perfunctorily. This is not enough and the correct toilet of the hands is a study in itself. But I would suggest that there should be a much more widespread use of gloves. They are cheap enough and if sterilization of them is a problem, they can be quite adequately sterilized by putting them on, washing them in soap and running water, wiping them over with an antiseptic of adequate strength and leaving it time to act before touching the wound. Lastly, may I point out that in view of the possibility that the clothing may be infected, the operator should be very careful to avoid touching his clothes.

We now come to the prevention of infection in hospital. I think we ought now to lay it down quite definitely that it is a criminal proceeding to nurse an infected case of any kind in the same ward as open clean wounds. I have myself encountered instances in which suppurating wounds were being nursed cheek by jowl with patients who had just had clean operations such as herniotomies. But in addition, every case of nasopharyngeal infection in a surgical ward should be immediately removed, isolated and examined bacteriologically and only when pathogenic strains of haemolytic streptococci are shown to be absent should it be returned to the ward. I know that all this may seriously disrupt hospital routine and will undoubtedly be very unpopular with matrons and superintendents, but in my opinion it is the only safe way.

Thus if we wish to keep wounds free from infection, we must be eternally vigilant to prevent the ingress of organisms over the whole of the period the

wound is open. And now that we know the principal sources of the organisms there is no particular reason why the appropriate measures should not be taken.

Treatment of the Wound

I have said very little about the treatment of the wound itself, principally because this is a surgical problem. There is, however, no doubt that it should receive a thorough surgical toilet as soon as possible after infliction. This should include cleansing, excision, débridement and the like. I am very doubtful whether antiseptics are of value in the treatment of any wounds. Secondly, the wound should be immobilised as far as possible by the use of plaster or suitable splinting. And thirdly, it should be dressed in such a way that the dressings need not be disturbed for long periods. All these measures will tend to diminish the risk of the ingress of haemolytic streptococci and give the tissues the best possible conditions for dealing with the organisms which may be present in the wound. This technique was, in principle, employed by Trueta in the Spanish Civil War and he certainly had very little sepsis.

Of the more specific methods for the prevention of sepsis, I will only refer to sulphanilamide and similar drugs given by mouth as soon after the infliction of the wound as possible. This has not been definitely shown to be successful in human beings but there is experimental evidence in its favour. Every casualty in the British Army is now given prophylactic doses of sulphanilamide for a period of 48 hours after wounding. The total dosage during that period amounts to 17 gm. The results of this measure are not yet available but it is highly probable that they will be encouraging.

In conclusion, may I ask you to reconsider the idea so firmly held by many of us that sepsis in war wounds and in civil injuries is inevitable. Too often we think that because a wound is ragged, dirty and full of foreign material, haemolytic streptococci are already present and that little or nothing can be done to prevent their development. If we would only recognize that these organisms, unlike all the others which commonly infect wounds, are almost certainly implanted afterwards, and possibly while the wound is being dressed, we might succeed in preventing a great deal of sepsis. And if I have persuaded some of you to think in this way, I shall be well content.

For a full bibliography on this subject the reader is referred to a previous paper by the author which appeared in the *Lancet*, 1940, vol. 1, p. 109.

The Whence and Whither of Milk Sanitation*

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IN any line of public health work it is well for us to stop occasionally and make a survey of the field to determine how far we have come, what we have accomplished, and what the future is likely to hold for us. Where the effort has been long continued, it requires real research and careful study if the past history is to be summarized satisfactorily. Where this cannot be done the best substitute is to secure someone who has participated in the work and ask him to reminisce. This is always a dangerous thing to do because men have a tendency to become garrulous under such circumstances.

Canadian workers took an active interest in and helped in the preparation of the first American Public Health Association Standard Methods of Milk Analysis Report issued in 1910. Dr. F. C. Harrison was a member of the Committee of which Dr. F. H. Slack, a brother of your Dr. A. J. Slack, was Chairman. Among others who have helped in this work are Dr. N. MacLeod Harris, Dr. A. L. MacNabb, and Dr. D. T. Fraser. Likewise, two Canadian workers, Dr. A. J. Slack and Mr. M. H. McCrady, are active and efficient members of the present A.P.H.A. Committee, and Dr. H. R. Thornton is serving as an Associate Referee.

In the University of Pennsylvania Archeological Museum there is a frieze secured by one of their expeditions to the site of old Babylon. This represents the dairy industry of the day. The milker is seated at the rear of the cow instead of at the side, a position which is that normally taken by those who milk sheep and goats. It is not recorded whether this method of milking cows was universally practised in those days. It is, however, evident that there were individuals at the time who objected to having visible sediment in their milk, for adjacent to the cow and the milker, a man is shown pouring milk through a vessel which appears to be a strainer. I doubt not that many a similar indication that people of old times liked clean milk could be found in the records.

However, we must admit that modern milk sanitation begins with the real development of bacteriology which began about 1880 when the gelatin and agar plate methods were developed. Four years previous Lord Lister had succeeded in obtaining a pure culture of *Streptococcus lactis* by the dilution technique. In the decade that followed 1880, solid plating media were generally used and before the end of the decade there was discussion over the question whether

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gelatin or agar was more satisfactory for use when determinations were made of the number of bacteria present in any given material. In recent years, Dr. A. P. Hitchens (1) has succeeded in determining for us just where Frau Hesse, the wife of Wilhelm Hesse, secured the idea that agar might make a satisfactory plating medium, and has shown us that the idea passed through Jersey City, New Jersey, in its passage from Java to Berlin.

It was this same Wilhelm Hesse, an assistant in Koch's laboratory, who began discussion as early as 1888 (2) of the best way to estimate numbers of bacteria in any given medium by using the agar plate method. In the early days it was thought that the agar plate that developed large numbers of small, regularly distributed colonies was the one that was most likely to yield an accurate count. It was not realized until later that overcrowded plates did not develop the full number of colonies that were possible. Hesse and Niedner's report (3) shows that it was rather generally agreed by 1898 that agar was better adapted for quantitative determinations than gelatin and soon after Hesse and Niedner (4) pointed out that the counting is more accurate and less tedious where there are not more than 100 colonies per plate. They felt that the plates should be held from 2 to 3 weeks in order to allow all possible colonies to develop. These same authors recommended the inversion of petri plates after hardening before they were put into the incubator, thereby reducing the possibility of the growth of spreading colonies. Later it was shown by Hill (5) and Breed and Dotterrer (6) that there was good reason in milk work for following our present practice of selecting plates so far as possible that develop more than 30 and less than 300 colonies per plate.

In spite of the fact that this technique developed first in Germany, it was really in the Boston area, or at least in the New England area, where the question was first raised whether it was necessary to allow enormous numbers of bacteria to develop in our fresh milk supplies before they reached the consumer. Our pioneer American dairy bacteriologist, Professor H. W. Conn, began writing papers (7) as early as 1890 discussing the source of the bacteria that were found in our milk supplies. He had a remarkably clear understanding of the relative importance of the various sources of the extraneous bacteria that found their way into milk supplies. Almost simultaneously, Professor Sedgwick (8) published a report on a bacteriological examination of Boston milk supplies and emphasized the need for better dairy sanitation.

As early as 1893 Dr. Coit of Montclair, New Jersey, induced Stephen Francisco, a dairyman, to undertake the production of an exceptionally clean milk for baby feeding produced under the supervision of medical men. This proved to be the beginning of certified milk. It is claimed that Montclair was the first city in America and probably in the world to undertake a regular bacteriological examination of its milk supply. Professor H. L. Russell (9) began his studies at Madison, Wisconsin, at about this time also. Dr. W. H. Park discussed the high bacterial count found in the New York City milk supply with suggestions regarding the sources of the bacteria as early as 1901 (10), and soon after Pro-

fessor F. C. Harrison and others began agitating the question of pure milk supplies in the Montreal area.

The milk regulation that really gave us the foundation of our modern approach to sanitation control of our milk supplies came from the New York City Board of Health. They introduced an ordinance in 1896 prohibiting the sale of milk in that city except under permit which was granted subject to rules and regulations of the Board. This method of control raised a storm of protest from dairymen, a natural reaction from an industry that had not previously been controlled. The regulations were challenged in the courts and the fight was even carried to the United States Supreme Court which rendered a decision ten years later supporting this fundamental regulation. From that time until this, the right of officially constituted Boards of Health to control the sanitary quality of municipal milk supplies through a licence system has never been successfully opposed. It is interesting to note that even in a recent legal decision where a business concern tried to secure an injunction in a Federal Court to prevent a Board of Health from enforcing a regulation of the Board claimed by the Board to be reasonable with claim supported by evidence, the decision was, that under these conditions the Board had a right to exercise its legislative prerogatives without restraint from the Court.

Another matter of interest is the fact that Boston was the first city of the Americas, and probably in the world, to fix a definite bacterial count limit for its milk supply (1905). Some may feel that the sky was the limit for the figure chosen for market milk as delivered was 500,000 per cc.

About 1900, responsible public health workers, organized in the Laboratory Section of the American Public Health Association, appointed a committee to standardize methods of making bacteriological examinations of water supplies. The Committee appointed did such an excellent job of formulating directions for carrying out the agar plate method and methods for detecting coliform organisms in water supplies in the first Standard Methods of Water Analysis Report issued in 1905 (11) that this report became a methods manual for bacteriological research for the following decade. It was not until 1915 that the Society of American Bacteriologists organized a separate committee for the development of methods for studying pure cultures of bacteria under the chairmanship of Dr. H. J. Conn, son of Professor H. W. Conn. Following this, the American Public Health Association reports became more what they were intended to be, reports outlining control procedures useful in routine work.

When the water report was first issued, it stirred up much interest because of its excellence for the period in which it was issued and a demand arose for a similar report to outline methods for the bacteriological examination of milk supplies. The Committee that undertook this carried out many studies in counting technique now buried in literature (12) rarely available to the modern dairy bacteriologist. Their work resulted in the presentation of the first edition of the Milk Report at Richmond, Virginia, in 1910 (13).

The Milk Committee had had much difficulty in deciding between various viewpoints regarding the significance of bacterial counts in milk control work

and their report shows some evidence that there was compromise in the Committee as two incubation procedures were recognized. Plates were to be incubated either for 48 hours at 37°C., or for 5 days at 21°C. The infusion agar proposed was one known at the time as the most suitable for growing the largest possible number of bacteria.

At that time and practically from then until now, there have been two schools of thought regarding the use that should be made of bacterial counts from milk supplies. One group felt that the important thing was to detect the total amount of bacterial life present in the milk. This group realized that the amount of germ life in it did not indicate whether the milk was safe for use, but they felt that if the total bacterial content was considered in connection with the age and temperature of the milk, it yielded an indication of the care which had accompanied the production and handling of the milk. It was freely recognized that counts from buttermilk or sour milk much in excess of those obtained from fresh milk did not in any way indicate that these milk products were unsafe for use.

The other viewpoint was maintained by a group who felt that the total count was a matter of minor importance because in itself it did not throw definite light upon the safety of the milk as a food for human consumption. This group believed that samples of milk as delivered to the consumer should be examined by methods that would reveal whether there were organisms present that would cause disease. They were uninterested in the use of a medium that grew harmless types of bacteria and wanted quick results, obtained by incubation at body temperature. Their thought was that pathogens would appear on plates incubated in this way. This group held ideas that paralleled those held by many water analysts from then until the present.

While the first group had primary control in the Committee that prepared the first edition of the Milk Report, the second group came to have control in the period between 1910 and 1914. In 1914 the nature of the original report was completely changed by the introduction of a beef extract agar in place of the beef infusion agar, and the elimination of incubation for 5 days at 21°C. This fundamental change was made in a one-page report of the Milk Committee adopted at Jacksonville, Florida, in December of 1914.

During the period between 1916, when this change was incorporated in a more extensive report which has been regarded as the second edition of the Milk Report, and 1934, the type of agar adopted in 1914 has remained in use with modifications, that have, at times, quite changed the nature of the counts obtained. A summary of the changes in the several editions of the report of the Committee on Standard Methods of Milk Analysis, from 1910 to 1939, is given in table I. During all of the period of these changes there was a continuous demand for an agar that would both grow the pathogens that are most commonly found in milk, and yield a total count that gave a more complete picture of the total bacterial flora present.

Meanwhile, in European countries, the thought that counts from milk supplies should really represent the total flora present dominated the picture in

TABLE I

STANDARD METHODS OF MILK ANALYSIS

Changes in Agar Medium and Temperature of Incubation in Various Editions.

Edition	Date	Nature of Agar Medium	Incubation temperature	Reaction
1	1910	Agar, 1%; Witte peptone, added to a beef infusion 1%; Formulas for lactose (or dextrose) agar and whey agar also given. Coliform technique given.	48 hrs. at 37° C. or 5 days at 21°C.	+1.5 Fuller's scale.
	1914	Changed to 1% peptone; .5% Leibig's beef extract when possible. (Am. J. Pub. Health, 1915, 5:68). Coliform technique dropped.	48 hrs. at 37° C.	+1.0 Fuller's scale.
2	1916	Agar 1.5%; best available peptone .5%; Liebig's beef extract if available .3%; added to water.	48 hrs. at 37° C.	Between +.5 and +1.0 Fuller's scale.
3	1921	Agar 1.5%; Witte peptone if available, Armour's, Digestive Ferments Co., Parke Davis Co., or nearly any good commercial peptone .5%; Liebig's beef extract where obtainable .3%; added to distilled water.	48 hrs. at 37.5° C.	pH 6.2 to 7.0. Optimum pH 6.5 to 6.6 but unadjusted if within limits given.
4	1923	Agar 1.5%; Witte peptone if available, Armour's, Digestive Ferments Co., Parke Davis Co., Fairchild's permitted .5%; Liebig's beef extract or comparable brand .3%; added to distilled water.	48 hrs. at 37.5° C.	pH 6.2 to 7.0 as above.
5	1929	Agar 1.5%; Bacto peptone or comparable brand .5%; Bacto beef extract or comparable brand .3%; added to distilled water.	48 hrs. at 37° C.	Do not adjust reaction if between pH 6.2 and 7.0. Preferred pH 6.6.
6	1934	Agar 1.5%; Bacto peptone or comparable brand .5%; Bacto beef extract or comparable brand .3%; added to distilled water. Coliform technique reintroduced.	48 hrs. at 37° C. Tolerance 35.5 to 37.5° C.	Do not adjust reaction if between pH 6.4 and 7.0. Preferred pH 6.6.
7	1939	Agar 1.5%; Tryptone .5%; Bacto beef extract .3%; Glucose .1%; Skim milk where dilutions are greater than 1: 10, 1%; added to distilled water.	48 hrs. at 37° C. Tolerance 35 to 37° C.	Reaction preferred pH 7.0 Tolerance pH 6.6 to 7.0.

practically all of the countries that developed an interest in milk sanitation. This was particularly demonstrated by the studies and report (14) made by Professor G. S. Wilson in London for the British Ministry of Health after a visit to the United States and Canada to study methods of milk control in use in North America. Similar action was taken in Germany at approximately the same time.

During the period following the World War there was much complaint that the nutritive qualities of the standard agar then in use were inferior. Because it was believed to be possible to develop an agar which was more easily prepared than infusion agar that was at the same time capable of growing practically all of the bacteria in milk, including possible pathogens, studies were undertaken about 1934 using newer types of peptone with the inclusion of small amounts of sugar and even milk. The primary purpose of adding the latter ingredient was to stabilize one of the uncontrolled irregularities that greatly affects counts obtained with media that do not contain milk. Many analysts had noted the fact that higher counts were secured from the same samples from higher dilutions. This irregularity is normally caused by the presence in the agar of nutrients from the milk introduced in the inoculum.

During the years that had passed since 1914, it had become very evident that the pathogenic organisms that have caused the greatest amount of difficulty when present in milk supplies did not grow on the standard agar then in use. The pathogenic organisms in question are the organisms of tuberculosis, undulant fever and septic sore throat. Only a limited number of pathogenic organisms were capable of growing on a standard agar which contained Bacto-peptone and meat extract only. Quite independently, the American Association of Medical Milk Commissions, under the leadership of Dr. J. Howard Brown, undertook a similar study with similar aims. The conclusions reached were similar to those reached by referees appointed by the Laboratory Section of the American Public Health Association. Because the American Public Health Association Committee (15) found in the final comparative testing that the formula that was finally adopted was slightly better than the American Association of Medical Milk Commission's formula, the medium finally adopted by the American Public Health Association in 1939 was the one now known as the new standard milk agar.

Various things had conspired meanwhile to make the situation more complicated. Several laboratory methods, such as the microscopic method, had been developed for counting bacteria and the errors in agar plate counts were better understood; the methylene blue technique had been developed as an indirect method of estimating bacterial populations and control officials had utilized a wide variety of applications of these various techniques in the improvement of municipal milk supplies. It was and is quite evident that various methods of improving milk quality may be used with success, pasteurization being recognized as the one thing that gives blanket protection against the presence of pathogenic bacteria (provided, of course, that the pasteurization is properly carried out and the pasteurized milk is protected from recontamination after pasteurization).

Hence, greater emphasis has been placed on controlled operation of pasteurization equipment and in the last few years laboratory methods, the Kay-Graham and other phosphatase tests, have been developed by which it is possible to determine whether milk has been properly pasteurized. These changed conditions have also led to the use of coliform tests of milk supplies for determining with a fair degree of certainty whether milk has been properly pasteurized and protected from recontamination.

The change from the type of agar that had been used in milk supply control up until 1939 was brought about after careful study had shown the effects that would be produced by the change in the nature of the agar. In connection with these studies, it became increasingly evident that no counts could be made with any real degree of accuracy if the incubation temperature was retained at 37°C. It was found that normal milk bacteria, including the types that appear on plates at 37°C., grow rapidly at 32°C. and that at this temperature variations in the temperature of incubation did not cause the serious discrepancies in counts produced when 37°C. incubation was used. It is evident that if both the new agar and a lowered incubation temperature were used the chief purpose of standardization, i.e., ability to duplicate analytical results, could be attained reasonably well. While 37°C. incubation only is recognized in the Seventh Edition of the Milk Report, the American Public Health Association Committee having the matter in charge has already decided to recommend the making of 32°C. incubation optional in the forthcoming Eighth Edition. Action on this matter will be taken at the Detroit meeting of the American Public Health Association in October.

What of the future? It is very difficult for anyone to prophesy in these days when things, both political and in the public health field, change so rapidly. It may interest you to know something of the thought of some of the progressively minded younger and well-trained men. About twelve years ago adequate appropriations were made by New York State for the development of a real state-wide supervision of milk control work outside of New York City. Deputy Commissioner of Health, Paul B. Brooks, took a real and constructive interest in this work, and Mr. W. D. Tiedeman was put in charge. Two mobile laboratories were put into operation and the personnel was selected under standards that really secured the selection of trained men. After eight years of experience with this group, Mr. Tiedeman summarized his views in regard to later trends in the laboratory control of the quality of milk supplies (16). In going from city to city to check the efficiency of the program carried out by local health officials the workers in the mobile laboratories found that they could determine the quality of a given milk supply much more effectively by the use of simpler laboratory procedures than by the making of agar plate counts.

Their way of doing this has been to collect samples of bottled milk as delivered, examine the milk by the phosphatase test, make a microscopic preparation and also examine it for the presence of coliform organisms. Where phosphatase tests give indication of under-pasteurization, rechecks are then made at the pasteurizing plants. Rechecks are also made when positive coliform tests are obtained. The microscopic examination served as a general check on the total

number of bacteria present, indicating as it does, the presence of psychrophilic bacteria, thermophilic bacteria or other bacteria that do not grow where the standard plating procedure is used with 37°C. incubation. If no bacteria are evident under the microscope, it is clear that the milk supply is not badly contaminated. Neither has the milk stood for any length of time at a warm temperature. Dead bacteria are just as significant in revealing this past history as living bacteria.

The tendency in New York State, as elsewhere, has been to reduce the number of so-called grades of milk based on agar plate counts to but a single grade. New York City is, at the moment, in the midst of a discussion over a proposal made by the Department of Health to recognize but one grade of milk after September 1, 1940. If, as is expected, this regulation stands without modification, it is expected that all cities in upstate New York that still have two grades of milk will follow with the adoption of regulations recognizing but a single grade of milk, i.e., the safest milk that can be provided for the community.

These developments in New York State have been discussed by the author not because they represent the only progressive thought in milk control work but because they are the ones most familiar to me.

Without question, the milk supply of the future will be pasteurized, and its safety will be maintained by the use of a certain amount of laboratory examination of the milk. The exact tests that will be used by public health workers may vary somewhat according to personal preferences, but it is clear that methods that yield results that enable the official to determine whether the milk has been properly pasteurized and protected against recontamination are likely to be regarded as fundamental in importance. There are many individuals who feel that there is no reason for attempting to reduce the number of bacteria in pasteurized milk supplies to a level below that where the microscopic technique loses its usefulness, so that I am not surprised at the increasing interest in the use of the microscopic method of examining pasteurized milk supplies.

Thus far we have merely discussed the question of the final safety of the milk as it reaches the consumer. The modern consumer is not satisfied merely with knowing that his food is free from disease germs. He also wants it to be clean and sanitary. Hence, the introduction of pasteurization has not really lessened the demand that our milk supplies be clean and sanitary before pasteurization. Dairy inspection has been found to be essential in this field because no laboratory method has been developed whereby the difference between "clean" and "cleaned" milk can be detected. It is only through personal knowledge that dairy practices are cleanly, stables and milk houses clean, that we can assure ourselves that certain features in milk sanitation have been observed.

On the other hand, rapid platform tests which make it possible to examine cans of milk as delivered at milk plants before they are dumped into the weigh vat are now held in high repute in the New York City and New York State work. Milk company inspectors as well as municipal inspectors are being required to detect and reject unsatisfactory milk before it reaches the consumer. More and more emphasis has been placed on these platform tests in the last

five years by Mr. Pincus, Mr. Abraham and their associates, who have charge of the control of the milk supply for New York City (17).

One test that can be applied on the platform by the trained worker with greater success than is generally believed is the odour test. New York City inspectors have been trained to detect odours that indicate that something is wrong with the quality of the milk as it is put on the platform. They likewise use strainer dippers to determine whether there is excess sediment or clotted milk in the can. Where there is any indication that the milk is unsatisfactory, it is rejected, samples being taken and given a microscopic examination on the spot. The microscopic examination provides additional information regarding the probable cause of the unsatisfactory nature of the milk. The entire record for any given sample gives the inspector a basis for judgment when he or the plant inspector makes a visit to the farm to correct unsatisfactory conditions.

At the present time, both New York City and New York State officials feel that they are carrying out a more satisfactory program for the control of the quality of the milk supplies sold in the City and State than they have ever used previously. From the standpoint of these officials there is very little use of the agar plate technique and it may be that this indicates a change that is likely to come more generally in the future.

There is good reason for insisting that raw milk be freed from undesirable types of bacteria, particularly disease germs, even though the milk is to be pasteurized. This purpose cannot be accomplished by merely keeping the number of bacteria at a minimum. Before disease germs can be eliminated, it is necessary to secure accurate knowledge of their nature and source, and then to devise methods of getting rid of them. This work has been largely accomplished in the United States so far as human and bovine tubercular germs are concerned. Work is also in progress through the area-plan and blood-testing of herds to eliminate Bang's disease in cattle. When this purpose is accomplished, undulant fever of man derived from bovine sources will disappear. Udder infections (mastitis) that cause the milk secretion to become abnormal are being controlled through physical examinations by veterinarians, laboratory examinations of milk and the better methods of herd management used by dairymen. Fortunately, the disease germ or germs that cause this very prevalent disease in cattle do not, so far as is known, cause human disease. A closely related human streptococcus infection that may be transferred to cattle under certain special conditions does cause epidemics of septic sore throat, scarlet fever, erysipelas and related diseases of human beings. The control of this type of disease germ has proved difficult, pasteurization being our only real safeguard against this germ.

Unfortunately, at the present time there is a tendency in the United States to spend an undue amount of energy and valuable time in getting some harmless types of bacteria out of our milk supplies. These bacteria are usually detected only because of the fact that they are difficult to kill during the pasteurization process. Efforts might better be concentrated on the extension of pasteurization as a blanket means of protection against milk-borne diseases and upon the

eradication of diseases that may be transmitted to human beings from our milk producing herds. A well-balanced milk inspection program will not only maintain the total number of bacteria in a milk supply below a certain reasonable minimum, but it will also result in the control or eradication of undesirable disease germs in the milk supply.

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Rabies Infection*

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IN Canada rabies in animals is a reportable disease and is subject to control by the Health of Animals Division of the Federal Department of Agriculture. The control and eradication of rabies have always been a difficult problem due largely to ignorance on the part of the general public and the general objection to the control of dogs. This ignorance is gradually being overcome but there is still sufficient misconception of the disease to render its control difficult in practice.

Rabies is an acute infection and almost always fatal. All warm-blooded animals are susceptible. The disease is characterized by nervous symptoms and mental disturbance and there is usually increased excitability followed by paralysis. Infection is nearly always transmitted by a bite and the causative agent is a neurotropic ultravisible virus.

The disease has been known since the time of Aristotle and was formerly believed to develop spontaneously. In 1804 Zinke demonstrated the infectivity of the saliva for dogs and it was later shown by different workers that herbivora and man became affected in the same manner. Pasteur and his co-workers in 1881-89 proved the virus was present in the central nervous system and with the inoculation of rabbits found it was possible to prepare a virus of known and constant virulence (fixed virus) which solved the problem of practical immunization in the human being. Remlinger and Riffat-Bey proved the filtrability of the virus in 1903 and Negri in the same year demonstrated specific bodies in the central nervous system of rabid animals. Negri bodies are absent on the average in only about ten per cent of cases. The definite relation between Negri bodies and the virus of the disease is not known. In herbivora and swine, Negri bodies are found less frequently than in dogs. The finding of these Negri bodies is still the main method of confirming the disease because examination can be made at once but in cases of doubt inoculation of rabbits is undertaken.

When any suspicion of rabies exists the dog should if possible be securely chained or enclosed where he cannot escape and allowed to die. If it is impossible to secure the dog he should be shot through the heart so that the brain is left intact for examination. When dogs die of rabies, Negri bodies are usually easily found but if the dog is destroyed in the early stages it may be impossible to find them. This necessitates the inoculation of rabbits and the delay of two to three weeks involved may be dangerous if persons have been bitten. Dogs usually die within four days after symptoms of rabies become evident. If death does not take place within a fortnight, rabies may usually be excluded.

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Rabies occurs in all parts of the world. Probably Great Britain and Australia are the only two countries entirely free from it. The disease usually appears in dogs, cats and wild carnivora (wolves, foxes, jackals, etc.), while other animals, and man, are less often affected. About 80 per cent of all cases of rabies originate from the bites of dogs. In man 90 per cent of cases originate from dogs.

A considerable effort has been made in recent years to control rabies in different countries. In countries where extensive outbreaks occur vaccination has been used and has undoubtedly reduced the infection considerably. Kondo, in Japan, reported that as the result of the inoculation of 1,685,265 dogs between 1918-1930 rabies occurred in 297 or 0.017 per cent, while 16,522 cases occurred amongst non-vaccinated dogs. In Budapest district in Hungary, 130,000 dogs were vaccinated in three years with marked success.

In the United States numerous districts have definitely reduced rabies as a result of vaccination but as the vaccination is usually arranged in conjunction with licensing of dogs the stray dogs have been a difficulty.

The vaccines used in the United States are either phenol or chloroform killed and if properly prepared contain no live virus. Generally a single inoculation is given. There has been considerable controversy over the efficacy of the single injection of rabies vaccine. It is generally recognized that a single injection does not give absolute immunity in all dogs, although when systematically carried out the number of cases of rabies in dogs is vastly reduced.

Before rabies vaccine is generally accepted its potency must be standardized and the possibility of artificial cultivation of the virus arouses hope that this may be accomplished. If multiple injections of the vaccines at present in use were practicable or increased doses could be used the effectiveness would, no doubt, be increased.

The United States Live Stock Sanitary Association Committee on Rabies reports: "Your Committee desires to state emphatically that the use of rabies vaccine alone, no matter how effective, cannot control disease, but that such vaccines can be used to advantage in some communities and under certain conditions in conjunction with other standard measures. The State-wide compulsory vaccination of dogs is not considered advisable as practised at the present time. Its compulsory use in restricted communities should be approached with caution and only on a sound basis of administration."

As a result of a questionnaire sent out by the United States Bureau of Animal Industry in May 1939 to Live Stock Sanitary and State Public Health Officers, there were reported for the year 1938—8,452 cases of rabies in dogs, 413 in cattle, 32 in horses, 164 in sheep, 42 in swine, 207 in cats, 11 in goats, miscellaneous 44 and in man 47, making a grand total of 9,412.

In Canada during the fiscal year ending March 31, 1940, an outbreak of rabies occurred in Ontario centred in Huron County in which thirteen cases were confirmed. These comprised 9 dogs, 1 cat and 3 cattle. In the district involved about 60 people received "Pasteur" treatment. To simplify control 22 townships in the counties of Huron, Bruce and Perth were constituted an infected place by Ministerial Order on October 25, 1939, and it was found possible

to remove the restrictions from this area March 6, 1940. The disease appears to have been eradicated.

Rabies has never been prevalent in Canada and while some considerable outbreaks have occurred in Ontario and Quebec and been eradicated, there have been long periods without a single case. There was no rabies in Canada in the fiscal years 1906, 1917-24 and 1936-1939, with the exception of an isolated case in 1938 in Toronto. This dog was in a cage when symptoms appeared.

CONTROL MEASURES

The most effective preventive action against rabies is the licensing of all dogs and destruction of stray dogs. If this action were conscientiously carried out by local authorities the risk of serious outbreaks would be reduced. In Canada the method followed when an outbreak occurs is to destroy known contact dogs which have been bitten and to quarantine all suspected contacts. The course which the rabid dog has followed is traced as far as possible and all dogs on the route quarantined. Dogs which are only possible contacts are held under quarantine for three months. Any which are more probable contacts may be held for six months. If, however, the number of dogs infected increases and there is difficulty in tracing their movements a Ministerial Order may be issued constituting several townships or counties an infected place from which dogs may be removed only under licence.

The co-operation of the local councils and medical health officers is always solicited and generally a ready response is received. Medical officers of health can influence their council to pass by-laws for the control of dogs so that their municipal officers can enforce them or if by-laws are already in existence they can encourage the council to have them enforced. Veterinary inspectors in turn are definitely instructed to report all cases where human beings have been bitten by rabid dogs or suspected dogs to the medical health officer, in order that measures may be taken to protect these persons by vaccination. With the success which has attended control in the above manner in Canada no change is anticipated in the regulations. In the countries or localities where rabies has become prevalent the use of vaccine as an aid to control is no doubt justified, but in Canada where every outbreak so far has been successfully controlled and the disease eradicated vaccination is not permitted.

The difficulties of absolute eradication may be appreciated from the outbreak which occurred in Montreal in 1927. The municipal authorities evidently did not maintain proper control over the dogs in the city. It became necessary for the Federal Government to detail officers with suitable equipment to catch dogs running on the streets and remove them to a suitable place for destruction. Although a muzzling order had been in force for nearly two years and due warning was given that all dogs found at large after a specified date would be destroyed over 2,500 dogs were caught and disposed of. After that drastic action the situation rapidly improved.

Since the dog is the source of rabies in this country, the problem is one for

the veterinarian. However, for sentimental reasons, based on the intimate relation of the dog to family life, control measures aimed at the eradication of rabies invariably meet with resistance from some sections of the public. For that reason and because of the danger to human life, the co-operation of public health officials and local authorities is essential for rapid eradication of rabies.

At the present time the rabies vaccines used for single inoculation are somewhat crude and unstandardized but a percentage of dogs are undoubtedly immunized. If a standardized potent product can be produced it would be a powerful factor as a control measure in districts where rabies is prevalent but it cannot be too strongly emphasized that vaccination, even if effective, will not eradicate rabies without control of stray dogs and quarantine measures.

The most important preventive measure is the elimination of the stray dog and this is best accomplished by the licensing of all dogs so that unclaimed stray dogs may be impounded and destroyed. As already mentioned, the Committee on Rabies of the United States Live Stock Sanitary Association has recommended that the control of rabies be placed in the hands of the Federal Government. If this is done there is no doubt that effective measures would be taken by the United States Bureau of Animal Industry and within a comparatively short time rabies would be within controllable limits.

The control of rabies in the United States is of importance to Canada since it is a reasonable assumption that outbreaks occurring in this country may originate from United States dogs. The disease has been eradicated in Canada on several occasions and if no outside infection were introduced the prospect of eliminating rabies from the list of diseases occurring in Canada would be quite promising.

Observations on the Nutritive Value of Bread

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A RECENT report (1) of a survey of Canadian families made by the Dominion Bureau of Statistics shows that while cereal products accounted for only 18 per cent of expenditures for foods they provided over 30 per cent of the total calories, almost 35 per cent of all protein, 11 per cent of calcium, and 25 per cent of iron and phosphorous in the complete diet. It is clear that cereal products, of which bread is the chief one, are important articles in the Canadian food supply. This is particularly true of low-income families; in spending a meagre food allowance most attention is generally given to foods which supply bulk at low cost. Records of food consumption by low-income families in Toronto have shown that individuals frequently eat twelve or more slices of bread a day.

While it has been recognized for years that much of the vitamin and mineral content of wheat is not present in white flour, white bread is still widely used. Despite extensive educational work only 20 per cent of the bread used in Toronto is whole wheat; the same percentage is likely true for all of Canada. The wheat grain is an excellent source of the members of the vitamin B complex; only about one-third of the total is found in white flour.

Persons interested in nutrition have frequently stated that there is a widespread deficiency of the B vitamins in diets customarily used. This is certainly true in the southern United States where pellagra and associated diseases have been a major problem for some years. Pellagra, beri-beri and other conditions resulting from a severe deficiency of the B vitamins do not occur to any extent in Canada, yet it is possible that many Canadians do not receive sufficient of these vitamins for health. In a survey of 100 low-income families by a Toronto committee (2), it was found that men were better fed than other members of the families, yet the men averaged 340 units of vitamin B_1 per day and 70 per cent of the men had less than this average. In a recent survey of Toronto families having incomes between $1,500 and $2,400, the men were found to receive an average intake of 390 units of vitamin B_1 per day, an amount not much greater than was found in the lower income group. A daily intake of 500 units of vitamin B_1 is considered to be satisfactory. Since the various members of the vitamin B complex generally occur in the same foods, it can be assumed that these individuals were securing insufficient amounts of all the B vitamins. The food habits of these individuals were not abnormal and the results found for them are likely true for many Canadians.

The intake of the B vitamins could be significantly increased by the use of whole wheat bread in place of white bread. There are, however, several practical

difficulties. The use of whole wheat bread has been widely urged for years but white bread is still preferred. The presence of bran in bread is not always desirable because of its irritating effect upon the gastro-intestinal tract. The percentage of whole-wheat flour in so-called whole-wheat bread varies widely. One baker may use 50 per cent, another 10 per cent. At present there is no guarantee that a purchaser of whole-wheat bread will secure the nutritive value which he assumes to be present. A definition of whole-wheat bread by the Department of Pensions and National Health is highly desirable. Only by such means can the consumer of bread be protected.

Because of the public desire for white bread, a number of suggestions and attempts have been made to restore to it part or all of the nutritive value lost in milling. Vitamin B_1 (thiamin) is readily available as a pure substance and the addition of it to white flour has been recommended. This would only supply one of the vitamins lost in milling. Attention has been focussed upon thiamin with a consequent unwise neglect of other B vitamins because thiamin is easily measured and because it is available commercially. Experimental evidence indicates that the B vitamins are jointly needed for metabolic processes and that they should be supplied in balanced amounts. It is difficult to see the wisdom of removing eight or more needed vitamins and then adding one only to make good the loss. Several methods are available to produce bread of good colour and texture, containing quantities of the B vitamins equivalent to those present in whole-wheat bread. A special high-vitamin yeast has been developed which can be used in making white bread of good vitamin content. Several millers have available flour containing wheat germ; such flours give bread which is almost white. Another simple plan is to add wheat germ to bread dough. In this case, also, the bread is almost white, has a good flavour, and does not contain bran.

Since there is evidence regarding the need for additional supplies of B vitamins in Canadian diets it seemed advisable to secure experimental data regarding the nutritive value of whole-wheat and white breads as commercially available and to study the effect of the addition of thiamin and of other supplements to white bread. Young white rats of the Wister strain reared in the Connaught Laboratories colony have been used. They have been housed in individual, screen-bottom cages with water continuously available. A group of ten rats was used for each experiment and the weight curves here presented are based on averaged weights for such groups. Rats for each group were chosen so that the average weight of the groups used in one experiment was equal at the start.

In the first series two groups were maintained on bread and water for 25 days, one group receiving white bread and the other bread containing 20 per cent whole-wheat flour. Each rat in this and subsequent experiments was given cod-liver oil twice a week. Weight curves are shown in figure 1 and the better growth produced by the whole-wheat bread is obvious. There was also a difference in appearance and behaviour of the animals. Those subsisting on white bread had coarse, matted fur and were listless, while the brown-bread rats had fur of normal appearance and were energetic. After 25 days each rat received a

daily supplement of 12.5 gamma of thiamin for 7 days. This caused no difference in the rate of growth, in behaviour, nor in appearance. Subsequently each animal was given dried brewers' yeast as 5 per cent of the diet. This supplied all the B vitamins and caused a marked change in rate of growth and in the condition of the animals. While the growth rate of the rats fed brown bread

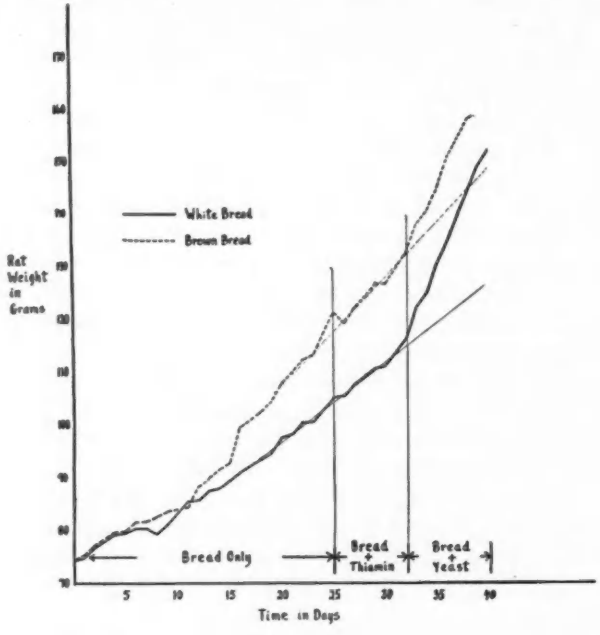

FIGURE 1

Growth rate of rats. Difference between white and brown bread. Lack of effect of thiamin. Value of B complex.

was increased, that of the white-bread animals was so altered that they became almost as heavy as the rats in the other group. Although thiamin alone had no apparent effect, the addition of the whole B complex to the white bread made it equivalent to whole-wheat bread in nutritive value.

Three groups of rats were used in the second series. One group received white bread plus 12.5 gamma of thiamin per rat per day. The second group was given a mixture of 95 per cent white bread and 5 per cent wheat germ and the third group was given only brown bread containing 20 per cent whole-wheat flour. Average weight curves are shown in figure 2. The mixture of white bread and wheat germ appeared to be equivalent to brown bread and both were definitely superior to white bread plus thiamin. It is again obvious that the addition of the whole B complex is much more desirable than the addition of only one vitamin.

Through the co-operation of a commercial bakery white bread containing

5 per cent wheat germ, added to the dough, was purchased. In the third series the nutritive value of this bread was compared with that of brown bread and of white bread. The average weight curves are shown in figure 3. At the end of 19 days rats which had received the wheat-germ bread were heavier than those given brown bread and much heavier than those given white bread. After that

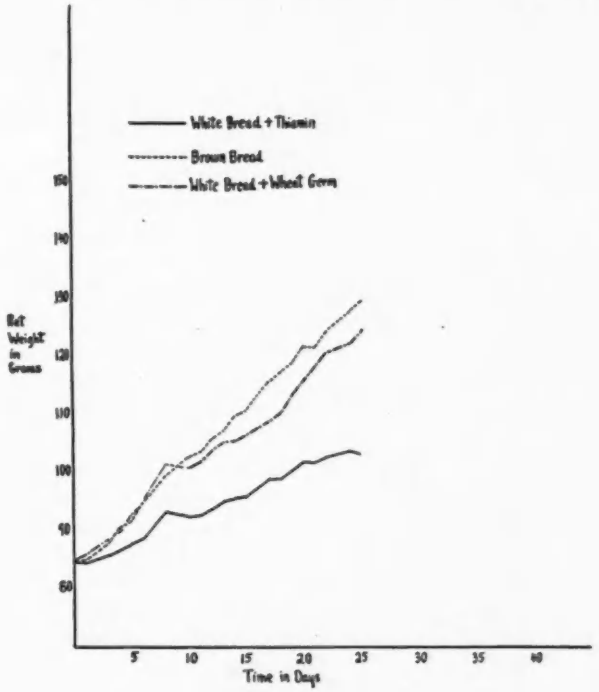

FIGURE 2

Growth rates of rats showing effects of brown bread, white bread and wheat germ in contrast with white bread plus thiamin.

time diets were changed. Animals previously receiving wheat-germ bread were given white bread; there was an immediate decrease in the growth rate. Both of the other groups were changed to wheat-germ bread and there was some increase in the animals previously receiving brown bread. The rats formerly given white bread responded to wheat-germ bread with a marked increase in the rate of growth. It is obvious that the bread containing 5 per cent wheat germ was superior to a commercial brown or white bread in its effect upon growth. The wheat germ bread was supplied to a number of humans and these all considered the flavour to be superior to that of white bread. From the viewpoint of palatability its use would cause no difficulty.

Nutrition is generally recognized as a factor influencing public health. If there exists in Canada a suboptimal intake of the B vitamins measures should

be undertaken to correct the deficiency. Wide-spread education has failed to make people like brown bread. Simple procedures are available to increase the nutritive value of white bread. For years, Canada has had a surplus of wheat. It would seem reasonable to improve the chief product of one of our largest industries so that it would be of greater benefit to the country. It is possible to

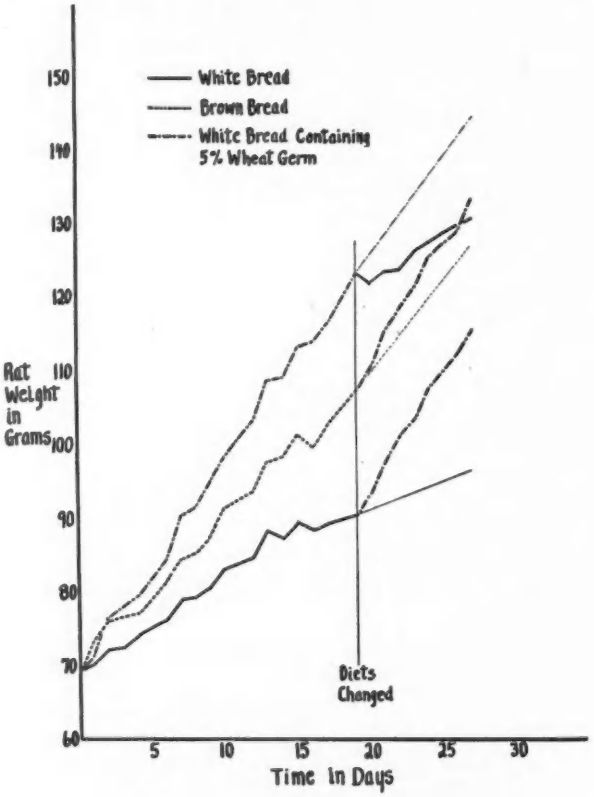

FIGURE 3

Growth rate of rats, showing nutritive value of white bread containing wheat germ.

give white bread the nutritive value of whole-wheat bread, without impairing the colour, by the use of high-vitamin yeast, or by adding wheat germ to the flour or to the dough. Any one of these procedures would improve nutrition. The adoption of such a plan should not be left to the initiative of the individual baker, nor to be exploited by him, but should be a matter of government regulation.

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Two Phage-Susceptible Types of B. Typhosus Isolated from a Typhoid Fever Case*

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c/o The University of British Columbia

CRAIGIE and Brandon (1) have reported a method of identifying V forms of *B. typhosus* by means of a specific Vi (type I) phage, while Craigie and Yen (2) have described how various types of the V form might be differentiated by using preparations of another Vi (type II) phage. With the help of Dr. Craigie, the Provincial Laboratories at Vancouver adopted these methods, nearly two years ago, as routine public health laboratory procedures. The highly specific action of the type I phage has permitted specimens yielding *B. typhosus* to be reported positive about 24 hours earlier than was formerly possible, and important epidemiological clues have frequently been furnished by type determination. This communication reports the distribution of the different phage types among 81 strains of *B. typhosus* isolated during the past 2½ years from patients living in various parts of British Columbia, and also records the facts relating to the simultaneous isolation of two V types (F_1 and F_2) from a single specimen of faeces from a case of typhoid fever.

Routine Procedure for Rapid Isolation of B. typhosus

The following procedure has proved highly satisfactory for the rapid isolation of *B. typhosus* from specimens of faeces and urine. The specimen is received in, or on arrival is immediately transferred to, a solution of 30 per cent glycerol in phosphate-buffered saline. Single plates of bismuth sulphite agar, Endo's agar, and eosin-methylene blue agar are streaked with faecal suspension, and one poured plate is also made with the bismuth sulphite agar medium, heavily inoculated with faecal suspension. After overnight incubation at 37°C., suspected colonies are picked, and transferred to nutrient broth, and to Russell's double sugar agar. If necessary, plates may be re-examined after 48, and again after 72 hours incubation, and suspected colonies picked. The broth cultures are incubated at 37°C. for about 4 hours, loopfulls from each culture being then transferred to a nutrient agar plate. After the inocula have dried,

*Presented at the eighth annual Christmas meeting of the Laboratory Section, Canadian Ppublic Health Association, Toronto, December 18-19, 1939, and subsequently revised.

drops of type I phage* are applied with a standard loop, according to the method of Craigie and Yen (2), to the centres of the inoculated areas on the plate, and the plates are incubated for about 6 hours at 37°C. Loopfulls from at least one of the broth cultures are similarly transferred to other nutrient agar plates, and are inoculated in series with the various type II phage preparations. The appearance of lysis at any area inoculated with type I phage may be taken to indicate that the culture placed there was *B. typhosus,* whose type will usually be revealed by the type II phage reactions on the other plates. Thus within 24 hours of receiving a faeces specimen, it may prove possible not only to report the presence of *B. typhosus,* but also to state the type. The phage-inoculated agar plates may alternatively be incubated for 2 hours at 37°C., placed in the refrigerator overnight, and re-incubated next morning for 4 to 6 hours at 37°C., when they may be examined and positive findings reported. The Russell's double sugar culture provides growth for slide agglutination tests, supplementary sugar reactions, and motility determination.

In the isolation of *B. typhosus* by blood culture, the blood clot is transferred to dextrose broth, and incubated at 37°C. when growth is apparent, a nutrient agar plate is streaked and incubated at 37°C., to provide a subsequent check upon the purity and identity of the culture. A plain broth tube is also inoculated from the original culture, and is tested, after 4 hours incubation at 37°C., for phage-susceptibility.

We have not yet encountered a strain showing typical confluent lysis with type I-IV phage mixture, used at the critical dilution, which subsequently failed to conform to the accepted tests for *B. typhosus.* However, on two occasions we have isolated late lactose-fermenting micro-organisms from faeces, both apparently non-pathogenic coliforms, which proved somewhat susceptible to the type I-IV phage mixture used at critical dilution. Although the frequency of occurrence of such strains could only be determined by extensive picking of colonies from stool culture plates, they are probably rare. But their significance became enhanced by our isolation of a new phage type of *B. typhosus,* provisionally designated type M, whose representatives have proved even less susceptible than these non-typhoid strains to the type I-IV phage mixture. While the 7 type M strains isolated to date have shown confluent lysis at critical dilution with a homologous type II phage, the type I-IV phage mixture used at its critical dilution produces with them only a slight uniform inhibition of growth over the inoculated areas of the plate, actual plaques being either sparse or altogether absent. An inexperienced worker might easily overlook such reactions. Apart from the occasional isolation from faeces of imperfect V, or even W forms, the existence of type M, and perhaps other relatively phage-resistant types, makes it undesirable for public health laboratories to rely solely upon phage-susceptibility reactions, and to dispense with the accepted confirmatory tests, for the identification of *B. typhosus.*

**In recent months, following a suggestion of Craigie (3), we have used a mixture of types I and IV Vi phages for identification of V forms, in place of the type I phage. The mixture gives more clear-cut results with strains of types A and E, which are less susceptible than other types to the type I phage, but are highly susceptible to type IV phage.*

TYPES OF B. TYPHOSUS ISOLATED IN BRITISH COLUMBIA

The distribution of the various types of *B. typhosus* among 81 consecutive strains isolated during the past 2½ years in the Provincial Laboratories at Vancouver is set forth in table I. At the outset, the strains were typed for us by Dr. Craigie, but for nearly two years now we have done our own typing. Duplicate cultures were frequently sent to Dr. Craigie for confirmatory tests, and whenever typing difficulties arose we had the benefit of his experience. It is noteworthy that 3 new types, viz. G and H (identified and designated by Dr. Craigie), and M (see above), should have been inaugurated by strains from among this comparatively small group of cultures from British Columbia. No

TABLE I

TYPES OF B. TYPHOSUS FOUND AMONG 81 BRITISH COLUMBIA STRAINS

TYPE OF B. TYPHOSUS	A	B_1	B_2	C	D_1	D_2	E_1	E_2	F_1	F_2	G	H	J	M	No typical V form	Total
No. of Strains.....	14	1	1	3	6	0	13	0	5	14	3	1	0	7	13	81

representative of these types was encountered by Craigie and Yen (4) among 418 strains from Quebec Province, or among 98 strains from England and Scandinavia. Of the 13 strains described in the table as having "no typical V form", 5 were actually of imperfect V form, of which only 3 (including 2 strains from carriers) had been recently isolated. Of 2 other strains of imperfect V form, and 8 strains of W form, all had probably suffered degradation through repeated subculture. Taking this into account, it proved possible to type 68, or 95.8 per cent, of 71 freshly isolated strains.

Type differentiation has on several occasions provided the local public health authorities with useful epidemiological evidence. With the exception of one outbreak, due to water apparently polluted with at least 2 types of *B. typhosus,* to which Craigie and Yen (4), and Brandon (5) have alluded, we have not isolated more than one type from any group of cases known to be epidemiologically related. Nor, until recently, had we encountered any instance of multiple specimens from the same patient yielding more than a single type of *B. typhosus.* Because no such instance appears to have been previously recorded, we have outlined below the laboratory findings relating to the isolation of types F_1 and F_2 *B. typhosus* from a case of typhoid fever. The clinical history, and the results of an epidemiological survey, are also briefly reviewed.

ISOLATION OF TWO TYPES OF *B. typhosus* FROM ONE PATIENT

Laboratory Findings

A blood specimen, and a specimen of faeces containing much fresh blood, taken from patient "X", were sent to the laboratory under the provisional diagnosis of typhoid fever. Following the procedures already outlined, the

blood clot yielded a culture resembling *B. typhosus,* from which three separate colonies were picked and transferred to broth. The specimen of faeces yielded numerous colonies resembling *B. typhosus* on the bismuth sulphite agar plate, of which nine were picked at random, and also transferred to broth. After 4 hours incubation at 37°C., the reactions of the various broth cultures to types I and II Vi phage were determined. All cultures showed confluent lysis with type I phage used in a dilution of 1×10^{-3}. Set up against a series of type II phages supplied by Dr. Craigie, and used at their critical test dilutions, strains derived from the three colonies from the blood culture plate, and from six of the nine colonies from the stool culture plate, showed complete lysis with phages F_1 and F_2. The remaining strains, derived from the other three colonies picked from the stool culture plate, showed no lysis with the type F_1 phage used at its critical test dilution, but showed confluent lysis with the type F_2 phage used at its critical test dilution. Since F_1 and F_2 types of *B. typhosus* are equally susceptible to type F_2 phage, while type F_1 phage in the higher dilutions will only lyse type F_1 strains, these findings pointed to the blood culture strains and six of the stool strains being of F_1 type, and to the other 3 stool culture strains being of F_2 type.

Confirmation of the identities of the various strains was obtained by two methods. First, nutrient agar plates were inoculated, each with a different one of the above strains, in the fashion prescribed for phage susceptibility tests. Serial dilutions of type F_1 phages, up to and beyond the critical test dilution, were applied to the inoculated areas on each plate, and after appropriate incubation, the degrees of lysis present were noted. To a similar series of plates, type F_2 phage dilutions were applied. Included in each series were plates inoculated with the F_1 and F_2 type-strains supplied by Dr. Craigie. Under these conditions, all the strains provisionally designated type F_1, reacted in strikingly concordant fashion to the F_1 and F_2 phages respectively. Moreover, their reactions matched that of the F_1 type-strain. Similarly, all 3 strains provisionally designated type F_2, matched the F_2 type-strain in their reactions to the F_1 and F_2 phages. These experiments were repeated several times, with consistent results.

The type identities of the strains in question were further verified by preparing type II phages, from single plaques, according to the method described by Craigie and Yen (2), from two of the three strains which had proved resistant to the F_1 phage. Type II phages were also prepared against several of the F_1 phage-susceptible strains. Table II shows the results obtained when a representative of each of these two groups of phage preparations was titrated against certain strains, selected at random for tabulation from among those actually tested. Phage II/T290c readily produced confluent lysis to the same high titre, when inoculated in serial dilutions on strains T290a and c, and on the type-strain F_1. On the other hand, the same phage preparation failed to produce more than a few plaques, even in relatively high concentrations, on strains T290h and k, and on the type-strain F_2. From this it can only be concluded that strains T290a and c are type F_1, and that strains T290h and k are

not type F_1. Again, phage II/T290k readily produced confluent lysis in high dilutions with all strains isolated from patient "X", and also with the F_1 and F_2 type-strains. Strains T290h and k are therefore of F_2 type. The laboratory evidence appears to show conclusively that both F_1 and F_2 types of *B. typhosus* were present in the faeces specimen submitted.

TABLE II

PHAGE REACTIONS OF STRAINS REPRESENTATIVE OF TWO TYPES OF B. TYPHOSUS (T290a, c, and T290h, k) ISOLATED FROM FAECES OF PATIENT X

PHAGE DESIGNATION	STRAIN	Dilutions of Phage							
		$1/10^2$	$1/5\times10^2$	$1/10^3$	$1/5\times10^3$	$1/10^4$	$1/5\times10^4$	$1/10^5$	$1/5\times10^5$
II/T290c	T290a	CL	CL	CL	CL	SCP	+n	+n	17n 9s
"	T290c	CL	CL	CL	CL	SCP	+n	+n	45(n&s)
"	F_1	CL	CL	CL	CL	SCP	+s	+s	38s
"	T290h	48n	11n	5s	5s	–	–	–	–
"	T290k	46n	12(n&s)	10(n&s)	2s	–	–	–	–
"	F_2	23(n&s)	4s	1s	2s	–	–	–	–
II/T290k.....	T290a	CL	CL	CL	CL	CL	SCP	+(n&s)	+(n&s)
"	T290c	CL	CL	CL	CL	CL	SCP	+(n&s)	+s
"	F_1	CL	CL	CL	CL	CL	+s	+s	+s
"	T290h	CL	CL	CL	CL	CL	+(n&s)	+(n&s)	+(n&s)
"	T290k	CL	CL	CL	CL	CL	+(n&s)	+(n&s)	+(n&s)
"	F_2	CL	CL	CL	CL	CL	+(n&s)	+(n&s)	+(n&s)

CL = confluent lysis; SCP = semi-confluent plaques; + = innumerable discrete plaques; 11n = eleven normal-sized plaques, 5s = five small-sized plaques, etc.
II/T290c = preparation of type II phage grown on strain T290c.
II/T290k = preparation of type II phage grown on strain T290k.

A second specimen of faeces obtained over six weeks later, eight days after the patient's discharge from hospital, yielded a total of 12 colonies on several plates, all giving type F_1 reactions.

Incidentally, the patient's blood serum showed no H or O agglutinins on several occasions between the first and twelfth week after onset of his illness, when tested against suspensions routinely used in the laboratory. However, some agglutination was obtained against suspensions of both the F_1 and F_2 strains isolated from the patient. This fact is mentioned merely to emphasize the importance of stool and blood culture in the diagnosis of typhoid fever.

Clinical History

The patient was a middle-aged man, of Germano-Russian extraction, who worked as a labourer, mostly at lumbering. Following several days of prodomal symptoms, he was hospitalized with a typical severe attack of typhoid fever. On the eighth day after admission to hospital, he had a severe haemorrhage from the bowel. The first stool and blood specimens were taken next day and sent to the laboratory. The treatment was symptomatic except for the administration of sulphanilamide throughout the illness. At the time of collection of the first stool specimen, he had taken a total of about 220 grains of sulphanilamide over a

period of eight days, while throughout the 37-day course of the acute phase of his illness 1,140 grains of sulphanilamide were administered. Convalescence was uneventful, and the patient was discharged after six weeks in hospital.

Epidemiological Data

A milk-borne outbreak of type F_2 typhoid fever, involving 34 persons, occurred about one year prior to patient X's illness, in a small community over 100 miles distant from his home. The patient had not visited this community for some years. Of the 14 F_2 strains listed in table I, 13 were isolated from patients involved in this outbreak, the remaining strain coming from patient X. No other F_2 strains have reached our laboratory since typing was begun two years ago. All 5 F_1 strains listed in table I (which include the strain isolated from patient X) have reached us since the above outbreak, but have come from scattered and apparently unrelated sources in the Province. For several weeks prior to the onset of patient X's illness, no F_1 strain had reached us from any source in British Columbia*; nor had any cases of typhoid fever been diagnosed in the small town in whose poorer section the patient lived. A few weeks after his illness began, *B. typhosus* was isolated from another case in the same town; but the strain proved to be of type B_2.

Careful enquiry failed to disclose any history of recent contact with a known case or carrier of typhoid fever. For at least seven years, no cases traceable to an intra-municipal source had occurred in the town, which is a prosperous agricultural centre. However, every year an average of two cases of typhoid enter the local hospital. These cases usually occur in spring and early summer, and have been traceable to a reclaimed area some miles from the town, which is often flooded in spring. Patient X's illness occurred in autumn.

He lived in a two-roomed shack with his wife and three adolescent children, none of whom had been sick for two years prior to his illness, or had ever knowingly suffered from typhoid fever. The water supply was piped from the municipal water works, but a common privy was shared with the occupants of three adjoining shacks. None of these neighbours had recently been ill, and there was no history of typhoid fever among them.

Analysis of his activities during the forty days immediately preceding the onset of his illness disclosed many potential opportunities, but no actual evidence, of carrier contacts. Between the fortieth and twenty-fifth days, he worked and lived at a lumber camp, in contact with 33 people, of whom 18 were Chinese. Between the twenty-fourth and eleventh days, he picked hops in company with 300 workers; but he lived at home during this period, and carried his own food and drink to work each day. On the ninth and sixth days before his illness began, he ate at a roadside store, his meals consisting of Canadian factory cheese, creamery butter, home-made bread, and tea drunk without milk. On the intervening days he lived at home and took his meals there. On the evening of the sixth day, he returned to the same lumber camp, where he remained until the

**Nor has any F_1 or F_2 strain been isolated in British Columbia in the eleven months elapsed since this patient's strains were identified.*

onset of symptoms. The sanitary conditions at the lumber camp were poor, but those at the hops fields were good; and as far as could be ascertained, no illness resembling typhoid fever had occurred among the workers at either place.

COMMENTS

The relatively small group of 68 British Columbia strains of V forms of *B. typhosus* which have so far been typed have fallen into 11 different types; whereas 356 V forms from Quebec Province, typed by Craigie and Yen (4), were distributed among only 7 types. The extent to which this difference may be attributable to the more heterogeneous racial origins of the population of British Columbia must, for the present, remain conjectural.

Two alternative explanations present themselves to account for the simultaneous isolation, from a single specimen of faeces, of F_1 and F_2 types of *B. typhosus*. The first possibility is that the patient was infected with typhoid bacilli from two distinct sources. Although such a contingency would seem to be remote, especially in the absence of definite epidemiological clues, the patient's environment at home and at work during the weeks prior to his illness certainly provided many opportunities for carrier-borne infection. Craigie and Yen (4) state "infections where there has been evidence of an epidemiological relationship have yielded only a single type of *B. typhosus*", adducing as the only exception a water-borne outbreak in British Columbia, in which types A, C, D, E, G, and H were isolated from a group of 13 patients, all of whom apparently contracted their infection from polluted water. The water yielded types A and E of *B. typhosus*, and one imperfect V form. However, if a larger number of typhoid colonies isolated from the water had been typed, strains representative of other types might have been identified; while had phage-susceptibility tests been done on several colonies, picked from the cultures obtained from patients involved in the outbreak, instances of multiple type infections might have been detected. The apparent multiple infection of patient X was only detected because several colonies had been picked for typing.

The alternative possibility is that the patient was infected with type F_1, and that an *in vivo* change of type occurred to some of the F_1 micro-organisms, owing to an inherent type instability of the strain, or to exposure of the strain to the action of sulphanilamide, or perhaps to both factors. In an attempt to verify this possibility, some experiments were conducted in the Department of Bacteriology and Preventive Medicine, University of British Columbia, by Mr. F. W. Brason. Sulphanilamide was administered to rabbits and mice, which were subsequently infected, by various routes, with type F_1 *B. typhosus*. Although some reduction in phage-susceptibility appeared to occur in the inoculated strains under these conditions, even to the extent of some imperfect V form colonies being isolated from certain of the infected animals, no type F_2 colonies were identified.

SUMMARY

1. The action of Craigie's type I and II bacteriophages against V forms of

B. typhosus has been advantageously employed for 2½ years in routine public health laboratory practice. The method facilitates earlier identification, and may furnish important epidemiological clues, but the established confirmatory tests for *B. typhosus* should not be omitted.

2. Of 81 consecutive strains typed, all of which were originally isolated from residents of British Columbia, 71 were freshly isolated. Of these, 68, or 95.8 per cent, could be typed. The remaining 3 strains were of imperfect V form.

3. The relatively small group of 68 British Columbia strains of V forms of *B. typhosus* fell into no less than 11 different types; and furnished 3 hitherto unknown types. One of these, type M, has proved relatively resistant to a type I-IV phage mixture at dilutions giving confluent lysis with the other V form strains so far encountered.

4. The simultaneous isolation, from a specimen of faeces, from a case of typhoid fever, of type F_1 and F_2 colonies of *B. typhosus,* is reported. There was no definite epidemiological evidence pointing to a double infection having been incurred.

ACKNOWLEDGMENTS

We desire to record our appreciation of the help and co-operation received from Dr. J. Craigie, without which it would have been impossible for us to undertake typing of *B. typhosus*. We wish also to thank Dr. A. R. Wilson of Chilliwack for his kindness in supplying the clinical history of his patient.

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IMPROVING THE STAFF OF LIFE

THROUGH many generations of human experience bread has been shown to be a valuable food. It is important to Canadians because it is cheap and because it is derived from our chief agricultural product. Until the introduction of white flour in the nineteenth century bread was made from whole grain flour and was much more nutritious than the white bread for which a strong preference has been created. Whole wheat is a good source of the B vitamins but two-thirds of the vitamin content is removed in making white flour. Humans are robbed of the vitamins they need and cattle foods are enriched at human expense by modern milling practice. Dietary surveys indicate that the intake of the B vitamins, particularly in low income groups, is not sufficient for health. A great deal of nutritional education has failed to shift public preference from white to brown bread. Under present conditions it is perhaps as well because brown bread may mean bread not much better than white bread in many cases. Bakers can use any percentage of whole wheat flour they wish. The public should be protected by a government regulation stipulating the minimal amount of whole wheat flour present in brown bread.

Public preference for white bread is so strong that suggestions have been made for its improvement. Vitamin B_1 is readily available as a synthetic product and the addition of it to white bread has been urged. Commercial interests have given the impression that the incorporation of vitamin B_1 into white bread would make it nearly equivalent to whole wheat bread. The experimental data given in a paper in this number of the Journal show that such is not the case; only by the addition of all the B vitamins can the desired result be secured. Great Britain has recently decided to make the addition of vitamin B_1 to bread compulsory. Criticism of this step is now appearing in the medical press and the Medical Research Council has wisely urged changes in milling processes to increase the content of all the B vitamins. The nutritive value of white bread could easily be increased without altering the colour and without increasing its cost to the consumer. As a simple measure of improving Canadian nutrition, public health authorities are urged to give consideration to this important problem.

Twenty-Ninth Annual Meeting

CANADIAN PUBLIC HEALTH ASSOCIATION

in conjunction with the Annual Meeting of the

MANITOBA MEDICAL ASSOCIATION

Fort Garry Hotel, WINNIPEG, September 19-21, 1940

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Directory of Sessions

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THURSDAY, SEPTEMBER 19

8.30 a.m. **REGISTRATION. Every member and visitor is asked to register.**

9.15 a.m. **General session, Canadian Public Health Association.** Macdonald Room.

12.30 p.m. **Executive Council, Canadian Public Health Association:** Luncheon and business session. Salon C.

2.00 p.m. **Joint session, Canadian Public Health Association and Manitoba Medical Association.** Concert Hall, Seventh Floor.

8.30 p.m. **Public meeting.** Auditorium Concert Hall.

FRIDAY, SEPTEMBER 20

9.00 a.m. **General session, Canadian Public Health Association.** Macdonald Room.

12.30 p.m. **Joint luncheon, Canadian Public Health Association and Manitoba Medical Association.** Concert Room. Tickets ($1.00) may be obtained at the Registration Desk.

2.00 p.m. **Joint session, Canadian Public Health Association and Manitoba Medical Association.** Concert Room.

7.30 p.m. **Joint dinner, Canadian Public Health Association and Manitoba Medical Association,** followed by a dance. Dining Room, Main Floor; Ball Room, Seventh Floor. Tickets ($2.50) may be obtained at the Registration Desk.

SATURDAY, SEPTEMBER 21

9.00 a.m. **General session, Canadian Public Health Association:** Symposium on the Preschool Child. Program arranged by the Public Health Nursing Section. Macdonald Room.

1.00 p.m. **Public Health Nursing Luncheon.** Arranged by the Public Health Nursing Section of the Manitoba Association of Registered Nurses.

Program

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THURSDAY, SEPTEMBER 19, 8.30 A.M.

Registration: Every member and visitor is asked to register. There is no registration fee.

THURSDAY, SEPTEMBER 19, 9.15 A.M.

GENERAL SESSION, CANADIAN PUBLIC HEALTH ASSOCIATION

Macdonald Room

Chairman: DR. R. O. DAVISON, Deputy Minister of Public Health, Province of Saskatchewan, and President, CanadianPublic Health Associatino.

Address of welcome by the Minister of Health and Public Health, Province of Manitoba, the Hon. I. B. GRIFFITHS; and Chairman of the Health Committee, City of Winnipeg, Alderman P. BARDAL.

1. **The St. James - St. Vital Health Unit, Manitoba.** DR. I. M. CLEGHORN, Medical Director.
2. **Industrial Health and National Defence.** DR. J. G. CUNNINGHAM, Director, Division of Industrial Hygiene, Department of Health of Ontario, Toronto.
3. **Developments in Public Health in Montreal during the Past Three Years.** DR. AD. GROULX, Director, Department of Health of Montreal.
4. **Sanitary Supervision of Milk in Winnipeg.** DR. M. S. LOUGHEED, Medical Officer of Health, Winnipeg.
5. **The Present Status of Milk-borne Disease Hazards.** DR. C. E. DOLMAN, Director, Division of Laboratories, Provincial Board of Health of British Columbia, Vancouver.
6. **Progress in Pasteurization in Ontario.** DR. A. E. BERRY, Director, Division of Sanitary Engineering, Department of Health of Ontario, Toronto.
7. **Contagious Abortion of Cattle and Undulant Fever in Man.** DR. J. S. FULTON, Director, Animal Diseases Laboratory, University of Saskatchewan, Saskatoon.

THURSDAY, SEPTEMBER 19, 12.30 P.M.

EXECUTIVE COUNCIL, CANADIAN PUBLIC HEALTH ASSOCIATION

Luncheon and Business Session

Salon C

Reports of Committees.

THURSDAY, SEPTEMBER 19, 2.00 P.M.

JOINT SESSION, CANADIAN PUBLIC HEALTH ASSOCIATION AND MANITOBA MEDICAL ASSOCIATION

Concert Hall, Seventh Floor

Chairman: DR. J. A. FERRELL, Associate Director, International Health Division, The Rockefeller Foundation, New York.

Preliminary Report on Maternal Deaths arising from Maternal Mortality Survey and Pregnancy Survey. DR. F. W. JACKSON, Deputy Minister of Health and Public Welfare, Province of Manitoba.

Abortion. DR. J. D. MCQUEEN.

Toxaemia. DR. ROSS MITCHELL.

Intermission. Inspection of Exhibits.

Puerperal Sepsis. DR. BRIAN BEST.

Haemorrhage. DR. F. G. MCGUINNESS.

Accidents of Labor. DR. S. KOBRINSKY.

THURSDAY, SEPTEMBER 19, 8.30 P.M.

PUBLIC MEETING—AUDITORIUM CONCERT HALL

Chairman: MR. R. G. PERSSE, President of the Cancer Research Institute.

1. **Dr. Harold Wookey, Toronto: Cancer and Its Control.**
2. **Dr. Donald H. Williams, Vancouver: Progress in the Control of Venereal Disease.**

FRIDAY, SEPTEMBER 20, 9.00 A.M.

GENERAL SESSION, CANADIAN PUBLIC HEALTH ASSOCIATION

Macdonald Room

Chairman: DR. R. O. DAVISON, Deputy Minister of Public Health, Province of Saskatchewan, and President, Canadian Public Health Association.

1. **Typhoid Fever Epidemic in St. Boniface.** DR. MAXWELL BOWMAN, Department of Health and Public Welfare, Province of Manitoba.
2. **Surveys of Rocky Mountain Spotted Fever and Sylvatic Plague in Canada.** DR. R. J. GIBBONS, In charge of the Laboratory of the Department of Pensions and National Health at Kamloops, B.C.
3. (a) **Clinical Aspects of an Epidemic of Human Encephalomyelitis in Saskatchewan in 1938.** DR. URBAN GAREAU, Regina.

   (b) **Relation of Equine Encephalomyelitis to the Epidemic of Human Encephalomyelitis in Saskatchewan in 1938.** DR. J. S. FULTON, Director, Animal Diseases Laboratory, University of Saskatchewan, Saskatoon.

4. **Presidential Address.** DR. R. O. DAVISON.
5. **The Protection of Unavoidably Exposed Persons against Tuberculosis.** DR. R. G. FERGUSON, Director of Medical Services and General Superintendent, Saskatchewan Anti-Tuberculosis League, Fort San.
6. **Progress in Public Health in the Province of Quebec.** DR. JEAN GREGOIRE, Deputy Minister, Ministry of Health of Quebec.
7. **Mental Hygiene as an Integral Part of a School Health Program.** DR. STEWART MURRAY, Senior Medical Health Officer, Metropolitan Health Committee of Vancouver.

FRIDAY, SEPTEMBER 20, 12.30 P.M.

JOINT LUNCHEON, CANADIAN PUBLIC HEALTH ASSOCIATION AND MANITOBA MEDICAL ASSOCIATION

Concert Room

Tickets ($1.00) may be obtained at the Registration Desk.

A feature of this session will be the presentation of the awards in the 1939 Canadian Rural Health Conservation Contest, conducted by the Canadian Public Health Association in co-operation with the American Public Health Association:

FIRST AWARD: St. James - St. Vital Health Unit, Manitoba.

AWARDS OF MERIT: Terrebonne County Health Unit, St. Jerome, Que.
Foothills Full-time Health District, High River, Alta.
Nicolet County Health Unit, Nicolet, Que.
Laviolette County Health Unit, Grand'Mère, Que.

The awards will be presented by DR. JAMES WALLACE, Associate Field Director of the American Public Health Association, New York.

FRIDAY, SEPTEMBER 20, 2.00 P.M.

JOINT SESSION, CANADIAN PUBLIC HEALTH ASSOCIATION AND MANITOBA MEDICAL ASSOCIATION

Concert Room

Chairman: DR. W. E. CAMPBELL, President, Manitoba Medical Association.

2 - 3 p.m. MEDICAL AND SURGICAL ASPECTS OF DUODENAL ULCER.

The Medical Treatment of Duodenal Ulcer. DR. DUNCAN GRAHAM, Toronto.

Present-Day Views on the Surgical Treatment of Duodenal Ulcer. DR. P. H. T. THORLAKSON.

Intermission. Inspection of Exhibits.

3 - 5 p.m. CHILD HYGIENE:

1. **What of the Canadian Child?** DR. ERNEST COUTURE, Director, Division of Child and Maternal Hygiene, Department of Pensions and National Health, Ottawa.
2. **The Nutrition Problem in the Child Health Program.** MISS ANNA SPEERS, M.A., Nutritionist, Children's Hospital, Winnipeg.
3. **The Prophylaxis and Therapy of Whooping Cough.** DR. HARRY MEDOVY, Winnipeg.

FRIDAY, SEPTEMBER 20, 7.30 P.M.

JOINT DINNER AND DANCE, CANADIAN PUBLIC HEALTH ASSOCIATION AND MANITOBA MEDICAL ASSOCIATION

Dining Room, Main Floor; Ball Room, Seventh Floor

Tickets ($2.50) may be obtained at the Registration Desk.

A feature of the dinner will be the presentation of honorary life membership in the Canadian Public Health Association to DR. E. W. MONTGOMERY, Professor Emeritus in Medicine, University of Manitoba, and formerly Minister of Health and Public Welfare, Province of Manitoba, and to the Hon. J. M. UHRICH, M.B., Minister of Public Health and Provincial Secretary, Province of Saskatchewan, and Honorary President of the Canadian Public Health Association. There will be no addresses.

SATURDAY, SEPTEMBER 21, 9.00 A.M.

CANADIAN PUBLIC HEALTH ASSOCIATION

Macdonald Room

Symposium on the Preschool Child
Arranged by the Public Health Nursing Section
of the Canadian Public Health Association.

Chairman: MISS ELIZABETH A. RUSSELL, Director of Public Health Nursing, Department of Health and Public Welfare of Manitoba, and Chairman of the Public Health Nursing Section, Canadian Public Health Association.

9.00 a.m. PROFESSOR H. R. LOWE, Superintendent of Education, Manitoba—**The Emotional Development of the Preschool Child.**

9.30 a.m. **A discussion** led by MISS K. RICHARDSON, MISS IDELL ROBINSON, and MISS CATES of the Winnipeg Nursery Schools.

10.00 a.m.—DR. O. J. DAY—**The Health of the Preschool Child.**

10.30 a.m.—Question Period.

11.00 a.m.—Business Session.

SATURDAY, SEPTEMBER 21, 1.00 P.M.

PUBLIC HEALTH NURSING LUNCHEON

Arranged by the Public Health Nursing Section of the
Manitoba Association of Registered Nurses.

Ladies' Program

THURSDAY, SEPTEMBER 19th

3.30 p.m. Tea at the residence of Mrs. W. E. Campbell, 246 Waverley Street.

FRIDAY, SEPTEMBER 20th

7.30 p.m. Annual Dinner and Dance, Fort Garry Hotel.

It is requested that wives of visiting doctors get in touch with our Registration Committee, located at the Fort Garry Hotel, upon arrival.

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Ladies' Committee

Mrs. George Brock
Mrs. W. G. Campbell
Mrs. A. M. Goodwin
Mrs. S. G. Herbert
Mrs. F. W. Jackson
Mrs. H. D. Kitchen
Mrs. H. Medovy
Mrs. H. Morse
Mrs. E. W. Stewart
Mrs. Digby Wheeler

Mrs. W. E. Campbell

THE ASSOCIATION'S WORK DURING 1939-40

(Part IV)

SEVENTH ANNUAL REPORT OF THE COMMITTEE ON CERTIFICATION OF CAUSES OF DEATH

IN its more recent reports, the Committee has directed its interest particularly toward the response of the medical profession to the use of the new medical certificate of death introduced in Canada in 1935. The Committee adopted this policy because it felt that it could serve the Dominion Bureau of Statistics and the Provincial Registrars-General to a significant degree by ascertaining what influence the new procedure in certification was having on statements of cause of death. This is important in respect to the relationship between the certifying physician's preference as to the cause of death for statistical purposes as indicated on the certificate and the choice of cause of death which would be made according to the rules of selection now in force in Canada. Other activities of the Committee include the instruction of medical students and physicians in the principles governing death certification, confidential certification and stillbirth registration and certification.

Section I: Education of Medical Students in the Principles and Practice of Death Certification

In 1937 this Committee prepared an Exercise on Death Certification which was printed by the Association and made available for teaching purposes to all medical schools in Canada. The exercise was subsequently translated into French for use in French-speaking universities. Over 1400 copies have been distributed since this project was initiated.

This method of approach toward improving the accuracy and the practical clinical value of cause-of-death statistics is probably the only satisfactory means at our disposal. Recognition of the importance of such a plan as is being thus sponsored by the Association was reflected in the recommendations of the Conference revising the International List of Causes of Death in 1929 and again in 1938. Registrars and vital statisticians throughout Canada continue to provide presumptive evidence that the efforts made to date have achieved considerable success—success which is reflected partly in the fact that the medical certificates completed by the younger physicians tend to offer considerable less in the way of difficulty or doubt than others.

This year the Committee conducted an inquiry of the medical schools employing the student exercise with a view of securing comments on its present form and any suggestions for modification based on teaching experience. The response to the inquiry indicated that the Exercise has proved an invaluable aid for teaching purposes and that the medical students appreciate, and are definitely interested in, practical instruction of this type.

At the time the Exercise is reprinted, it is planned to incorporate certain

revisions based on the suggestions made in response to the Committee's inquiries. Additional examples will serve to stress the importance of filling in the section dealing with deaths from external causes and to show how a *number* may be entered on the medical certificate in cases in which the precise clinical statement might not be desired or desirable (syphilis or gonorrhoea). A few "completed" certificates could be added to emphasize the correct procedure on the one hand and some of the common errors in certification on the other. The Committee might also provide a statement of correct procedure as a basis for uniform treatment of the examples which form the basis of the Exercise.

The inclusion by the Dominion Bureau of Statistics in any published revision of its Handbook on Death Registration, of sections on the use of the medical certificate and the avoidance of undesirable terms would be a further valuable step toward the education of physicians in death certification and the elimination of some of the defects still present. The teaching value of the Handbook published in 1936 was substantial and the Committee takes this opportunity of urging the Dominion Bureau of Statistics to reprint this document with appropriate revision and extension at an early date.

SECTION II: STILLBIRTH REGISTRATION

With the exception of the Province of Brititsh Columbia, single registration of stillbirths is now an established procedure throughout Canada. This circumstance is gratifying to the members of, and the contributors to, the work of the Subcommittee on Stillbirth Registration and Certification.

The reported favourable response which has attended the introduction of the new certificate suggests that considerable clinical information of definite value will be secured in this way over a period of years. There are many other problems with which this Subcommittee will now have to concern itself. Of these, the principal one at the moment is that of a simple workable classification of the causes of stillbirth. Some studies have already been made in this field by the Committee and its members. Other related problems include definition of terms, rules of selection where joint causes are stated, etc. Statistical practice in relation to the disposition of foetuses or infants born alive under twenty-eight weeks' gestation, also remains to be dealt with.

The pursuit of these problems is justified on clinical grounds alone. Three per cent of all births are those of stillborn foetuses. There need be no doubt about the need for the continued efforts of this Subcommittee.

SECTION III: CONFIDENTIAL CERTIFICATION

The Subcommittee on the Confidential Death Certificate has made considerable progress during the past two years. Dr. Paul Parrot, chairman of this Committee, conducted comprehensive inquiries in the Province of Quebec, the results of which have been published in the JOURNAL (1939, **30**: 335-342). These inquiries followed a special study of existing defects in certification.

At the last annual meeting, representatives of the leading life insurance

companies were present to discuss this question. The need of these bodies for access to the official medical statements was stated to be twofold: (a) in order to continue their contributions to vital statistics and public health, and (b) in order to protect the public and policy holders by checking fraud where such is suspected. There appeared to be no problem in Quebec concerning the securing of copies of the medical certificate, medical certificates being secured by the companies directly from the physicians concerned.

A special meeting of the Committee on the Confidential Death Certificate was held Monday afternoon, June 12th, at which were present: Dr. P. S. Campbell, Dr. H. E. Young, Dr. M. R. Bow, Dr. F. W. Jackson, Dr. R. D. Defries, Mr. T. E. Ashton, Dr. L. A. Pequegnat, Dr. M. A. Ross, Mr. G. L. Holmes, Mr. G. H. VanBuren, Dr. R. O. Davison, Mr. S. J. Manchester, Dr. H. C. Cruikshank, and Dr. A. H. Sellers. Discussion of the extension of the work of the Committee into other provinces resulted in the appointment of a small working group, consisting of Mr. T. E. Ashton, Dr. N. R. Rawson, Dr. Paul Parrot, Mr. S. J. Manchester, Dr. P. S. Campbell, Dr. H. C. Cruikshank, and Dr. A. H. Sellers. Four possible approaches were suggested:

(1) The use of a second certificate submitted to certain physicians.
(2) The use of specific inquiries concerning selected causes of death.
(3) The conduct of inquiries into present methods from the point of view of the physician.
(4) Replacement of the customary form with a confidential certificate in selected areas, as had been done in Quebec.

The basis of the whole problem was the known present weakness in vital statistics, but since we are ignorant of the *relative* part which various factors play in the deficiencies and difficulties which exist (ignorance on the part of physicians as to what is desired, lack of familiarity of physicians with the method of recording causes of death, desire to avoid statements of approbrious diseases, etc.), it was agreed that further experiments by the Committee required to be carefully planned and controlled. Any change involves a certain amount of education and this in itself influences physicians in the direction of improvement.

The conduct of further special inquiries by the Committee has had to be held in abeyance. However, in February 1940 confidential certification was generally introduced throughout the Province of Quebec. Here, recognized health departments or health units have access to the medical statements as in former practice. It is too soon yet to assess the ultimate nature and extent of the influence of this procedure on medical statements but the provision appears to be appreciated by both physicians and medical officers of health. A full year of experience in Quebec may throw some further light on the whole problem.

SECTION IV: CURRENT USE OF THE NEW MEDICAL CERTIFICATE OF DEATH

The Committee has already made two studies of the use of the new medical certificate of the cause of death, and its second such study, which appeared in the

fourth annual report of this Committee, suggested that the anticipated improvement was taking place. Reports from registration offices, however, continue to indicate that there is still room for improvement and that such improvement depends upon recognition by the physician of the principles involved in the use of the certificate and in stressing the importance of brevity in statements and the need of using the certificate as a place for recording the *opinion* of the clinician as to the cause to which each death should be attributed. Undoubtedly the publishing by the Dominion Bureau of Statistics of a Handbook on Death Registration and Certification incorporating the Fifth Decennial Revision of the International List of Causes of Death may provide an opportunity for again directing the attention of the medical profession to the basic needs and the fundamental requirements.

Recently, the Bureau of Statistics at Washington published a most comprehensive Handbook which admirably serves these objectives. Publication of this latter Handbook followed the preparation of a new standard certificate of registration of death by the Bureau of Census in January 1939; and this certificate, as well as the Handbook referred to above, are of interest to us in Canada because of the close similarity between the new American form, the Canadian medical certificate and the form recommended in 1925 by the League of Nations as a basis for international uniformity in this respect. At present, England and Wales, Northern Ireland, Australia, New Zealand, United States and Canada have a certificate of cause of death incorporating the essential principles of the form suggested as a basis for a medical certificate for international use. This is an important measure of progress. It is noteworthy that the new form of certificate introduced in England and Wales on July 1, 1927, was approved in almost identical form by the Commission Mixté de Statistique Sanitaire of the International Statistical Institute at Cairo in 1928.

The urgent need for some consideration of the whole question of statistical practice in the treatment of joint cause statements has been emphasized by the new medical certificate. A recent study of death certificates on which diabetes was mentioned (CANADIAN PUBLIC HEALTH JOURNAL, 1939, **30**: 435-444) indicated that "under prevailing conditions of medical certification and statistical practice, the true picture of mortality from diabetes and its trend cannot be secured from official vital statistics reports." Most recorded diabetes deaths occur at advanced ages (40 per cent in the age group 70 years and over) when diabetes may be present but not the underlying cause of death. Application of the rules of preference results in the addition of many deaths to the total diabetes mortality, which physicians would not intend to be so recorded. At the same time it must be assumed, of course, by those responsible for the tabulations, other indications to the contrary being lacking, that diabetes was an important factor in the death if it is mentioned on the certificate.

These observations apply not only to diabetes but to other diseases which at present enjoy wide preference. A measure of the influence of rules of choice in such cases (diabetes, cancer, tuberculosis, etc.) was made clear by studies undertaken by the British Register Office in 1935.

While rules of preference, when two or more causes are given, are necessary until physicians thoroughly understand the use of the death certificate for presenting clearly their opinion as to the cause of death, classification based on the physician's opinion as to the cause of death as expressed in the form of the statement of the cause on the medical certificate of death, is the ultimate objective in the tabulation of vital statistics.

The Selection of a Single Cause for Tabulation when Multiple Causes are Stated

The question of the application of rules for the selection of a single cause for tabulation when multiple causes are stated, is one of importance in vital statistics because it has been shown that "serious variations between death rates in different countries arise from the lack of uniformity in rules of selection." The Manual of the International List of Causes of Death, recently published by the British Register Office, designates three requisites to the securing of international comparability in cause of death statistics. These are:

(1) The adoption of a death certificate which provides for a separation of those causes which are important contributory factors but not related to the disease causing death and a statement in the proper sequence of the series of causes which led up to the immediate cause.

(2) The selection for tabulation as the cause of death of that cause shown *on the certificate itself* as being the condition which "initiated the train of events leading to death."

(3) The periodic publication of supplementary tabulations showing the total frequency with which each morbid condition is *mentioned* on death certificates.

This Committee is in agreement with 1 and 2 but not with 3. Its attitude in the latter connection is that the tabulations suggested are dependent upon the degree of detail which physicians in different parts of the country and in different countries customarily employ. Such a variation is independent of the frequency with which complicating morbid processes occur and this is a weakness in such tabulations which has already been revealed by studies made by the Committee. The frequency of multiple statements has been shown to be much higher in Canada and the United States than in Great Britain.

In England and Wales the established system of rules has been adhered to until the medical profession became accustomed to the new form of certificate, and the only variation in method of selection following the introduction of the new certificate was that the time sequence on the certificate was occasionally used to choose between two diseases of equally high preference or between two local diseases. A study of a random sample of death certificates in 1935, referred to by this Committee in earlier reports, showed that:

"instances of obvious misunderstanding of the proper use of the form had become comparatively rare and that there was no longer any need to delay the adoption of a method of selection based on the certifier's opinion as to priority of causes expressed by his or her arrangement of them on the certificate. The study also indicated, however, that important changes in assignment of deaths, and therefore in death rates, would result from such a change of method, and arrangements were consequently made for a dual assignment of deaths by the old and the new methods of selection during the period 1936-39, by means of which corrective factors (conversion ratios) could be calculated to ensure statistical con-

tinuity of death rates. During the years 1936-38 assignment was made in accordance with the 1929 List and each method of selection; for the year 1939 assignment is being made by the 1929 List and the old system of selection of rules on the one hand, and by the 1938 List and the new system of selection on the other, the latter forming the basis for the published tabulations for the year 1940."

This Committee notes with interest the progress made in Great Britain. The new English Manual, recently issued, contains instructions for selective procedure on the new basis (i.e., in accordance with the arrangement of causes on the certificate).

Canadian developments in the direction reflected by English practice must await more generally satisfactory use of the new certificate and the conduct of further studies of the influence of any possible changes on recorded death rates. The Committee has no doubt as to the ultimate outcome but inadequate experience with the new form of medical certificate is available for any change to be made yet. Meanwhile it strongly urges that no deviation from established rules of practice should be made, otherwise it may be impossible at a later date to ascertain the precise correction factors which will be needed to preserve a measure of comparability when the change is made.

SECTION V: THE FIFTH DECENNIAL REVISION OF THE INTERNATIONAL LIST OF CAUSES OF DEATH

In its last report the Committee directed attention to the principal features of the 1938 revision of the International List of Causes of Death. During the past year new Manuals have been published for both Great Britain and Northern Ireland, and the United States. In addition, a new Physician's Handbook on Birth and Death Registration has been published by the Bureau of the Census, Washington, and this will serve to familiarize physicians with the new list. The latter document is supplied with an index to the detailed list—an index which "presents terms and expressions considered desirable for certifying causes of death, together with certain others less satisfactory but frequently reported on death certificates." The provision is a valuable addition to the practical use of such a Handbook for both the practitioner and for the teaching of medical students.

The new list is being employed in Great Britain and Northern Ireland and in the United States as from January 1st, 1940. The old list will continue in use in Canada for the year 1940 with the exception of a few changes in detail involving rubrics 13, 35, 36, 42, 43, 44, 45, 47, 66, 88, 94, 115, 137 and 177. It is contemplated that the new list will be introduced in 1941.

SECTION VI: CLASSIFICATION OF THE CAUSES OF SICKNESS

The contemplated work upon revision of the morbidity classification, approved by the Dominion Council of Health in June 1938, has not been possible pending the publication of the new Canadian Manual to the International List of Causes of Death. Meanwhile, however, considerable use has been made of

the tentative list and further experience in the classification of causes of sickness is thus being secured.

In connection with a study of hospital discharges in Ontario the Division of Medical Statistics of the Ontario Department of Health undertook the preparation of a special morbidity classification based on the International List of Causes of Death but which included 1300 separate entities. The coding arrangement under this scheme involves the use of four digits, the last of which is concerned with sub-divisions of separate diseases or conditions. On the basis of this detailed classification a study is being made of the frequency of causes under the various headings with a view to preparing a short simple list of causes which might serve needs of *hospitals* in periodic or annual tabulations of morbidity. It is believed on the basis of experience thus far that a list containing not more than about 300 rubrics would prove generally satisfactory. The fact that hospital experience differs so materially from general sickness experience makes it clear that the classification needs in the hospital field may differ considerably from the needs in general sickness records. However, both types of classification must for the sake of national uniformity be possible of comparison and summation and this merely requires some simple arrangement for the use of corresponding code numbers.

The members of the Committee are indebted to Dr. A. H. Sellers for the preparation of this report and for the time which he has so generously given to conducting the work of the Committee.

DR. R. D. DEFRIES, *Chairman*; MR. E. S. MACPHAIL, DR. M. R. BOW, DR. PAUL PARROT, MR. S. J. MANCHESTER, MR. T. E. ASHTON, DR. E. GAGNON, DR. C. W. MACMILLAN, DR. G. F. AMYOT, and DR. A. H. SELLERS, *Secretary*.

CURRENT HEALTH LITERATURE

These abstracts are intended to direct attention to articles that have appeared in other journals during the past month. Any of the journals referred to may be borrowed for three days or longer if desired. Address requests to the secretary of the Editorial Board.

Atebrin in the Treatment of Giardiasis (Lambiasis)

THIS paper gives a list of different drugs used for the treatment of giardiasis. Most of these have proved to be useless. B. Galli-Valerio in 1937 (Schweis. med. Wsch., p. 1181) first introduced atebrin for giardiasis.

The dosage advised by P. de Muro is the same as suggested by him for malaria in 1935 (Riv. malariol., *11*):

		Per day during 5-8 days	
Children,	0- 1 years.....	¼ tablet	(0.005)
"	1- 3 "	½ "	(0.075)
"	3- 6 " 1	"	(0.10)
"	6-10 " 2	tablets	(0.20)
"	10-12 " 2½	"	(0.25)
Adults.....	3	"	(0.30)

These doses were administered 2-3 times immediately after meals. The patients were kept under observation from 1 to 6 months, their stools being examined twice a week.

In one case a slight, yellow cutaneous and scleral coloration occurred during the treatment with atebrin which disappeared after one week. This was not from the damage of the lower cells but from an accumulation of akridin substances in the subcutaneous tissues. Atebrin given per mouth is more effective than subcutaneously.

Experiments in vitro showed that vegetative forms of *Giardia lamblia* died in a few minutes in a 1:200 solution of atebrin.

P. de Muro, Deutsche med. Wnschr., 1939, *65*: 262.

The Serological Types of Haemolytic Streptococci in Relation to the Epidemiology of Scarlet Fever and its Complications

THE paper records in detail the results of a systematic study of the serological types of haemolytic streptococci in scarlet fever and their clinical and epidemiological relationships with special reference to cross-infection. In all 1831 cases were studied including 471 with complications.

Statistical records of these scarlet fever cases are set forth. All were examined bacteriologically in an infectious diseases hospital in a period of over thirteen months. Out of 415 cases in which swab cultures were made from throat and nose on admission, both throat and nose swabs yielded haemolytic streptococci in 115 cases and nasal swab alone in 9 cases (2.5 per cent.). The total number of throat swabs giving positive results was 357. In only 2 cases were different types of haemolytic streptococci found in the throat and nose.

The method of culture of the various strains, the preparation of type specific anti-sera, the technique of typing and methods of isolating the haemolytic streptococci from cases have been fully described in detail. The serological types encountered in this survey are set forth and show a marked predominance of Type I in the early inquiry and of Type IV in a later inquiry.

The frequency of appearance of new types of haemolytic streptococci during the clinical course of the disease is mentioned. In numerous instances a new type appeared to replace the original type and in only 6 of a series of 25 cases did the original type persist throughout the period of residence in hospital. It was found that on the day any complication occurred in a scarlet fever case only a single type of haemolytic streptococci was found to be present in the throat or discharge, suggesting that the strain responsible for the complication is present before the complication becomes evident.

Of 455 cases with complications

studied bacteriologically, organisms other than haemolytic streptococci were found to be the cause in 34 instances. Two hundred and eighty were due to a type of haemolytic streptococci other than that with which the patient entered the hospital (51.5 per cent.), whereas only 92 were due to the same type (20.2 per cent.). In patients who had been two weeks resident in the hospital 90 per cent. of the complications were due to new types of streptococci. Finally the types of haemolytic streptococci and the severity of the scarlet fever produced have been correlated in 949 cases without complications.

H. L. de Waal, J. Hyg., 1940, *40*: 172.

Typhoid Fever occurring in Immunized Persons

THIS paper deals with an epidemic of typhoid fever in a hospital population, 90 per cent of which had been immunized against typhoid fever. The epidemic broke out in the military hospital at Vich, Spain, in the last week of April 1938. The hospital at this time had 1,700 patients and a staff personnel of 200. The source of infection was one of three water supplies of the hospital which had become contaminated from the overflow of an adjacent sewerage system. The population of the hospital became constant for a period of five weeks with the outbreak of the epidemic. Questioning revealed that about 10 per cent of the 1,900 members had never been vaccinated against typhoid fever while the remaining 90 per cent. had received two or more injections of typhoid vaccine from one year to three months prior to the epidemic.

In all, there were 147 cases of typhoid fever proved by Widal tests and positive cultures from the blood, urine or stools and in several cases from all three sources. Forty-nine of these patients had never been vaccinated against the disease, or approximately 25 per cent. of the 190 unprotected persons. The remaining 98 patients had been previously immunized, giving an incidence of 6 per cent. for the immunized members of the hospital. The total incidence of the hospital was about 7.5 per cent. The case fatality rate for the non-immunized group was 10.2 per cent. and for the immunized group, 4 per cent. Only 14.3 per cent. of the immunized patients gave a typical typhoid clinical picture. Bradycardia, splenomegaly and leukopaenia, in order of frequency, were the constant features noted clinically.

Barney Malbin, J.A.M.A., 1940, *115*: 33.

Tularaemia

THE reported incidence of tularaemia in the U.S.A. now exceeds 2000 cases annually with a mortality of approximately 5 to 7 per cent. The disease has been recognized in each of the 48 states. It has been reported, too, in 10 other countries. Twenty-four forms of American wild life have served as sources of infection in human cases, with wild rabbits and hares responsible in 90 per cent. of infections. Insect vectors play an important role, particularly horse flies, wood ticks and dog ticks. Scratches or bites by cats accounted for 13 cases. A water-borne epidemic has been reported from Russia where water rats were thought to be the source of contamination, while investigation of an outbreak of tularaemia in beaver led to the discovery of contaminated streams in Montana.

The majority of cases occur among hunters, vacationists, butchers, housewives and laboratory workers. The organism usually gains entrance through skin abrasions and it is recommended that rubber gloves should be worn in handling game, and disinfection precautions should also be taken. Infected meat requires thorough cooking before being considered safe. Legislation restricting the purchase and sale or handling of wild hares and rabbits has been enacted in at least three states.

U.S. Pub. Health Rep., 1940, *55*: 667.